DRAFT
TOXICOLOGICAL PROFILE FOR
CESIUM

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

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UPDATE STATEMENT

Toxicological profiles are revised and republished as necessary, but no less than once every three years. For information regarding the update status of previously released profiles, contact ATSDR at:

Agency for Toxic Substances and Disease Registry
Division of Toxicology/Toxicology Information Branch
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

FOREWORD

This toxicological profile is prepared in accordance with guidelines developed by the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA). The original guidelines were published in the *Federal Register* on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile succinctly characterizes the toxicologic and adverse health effects information for the hazardous substance described therein. Each peer-reviewed profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but is described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

The focus of the profiles is on health and toxicologic information; therefore, each toxicological profile begins with a public health statement that describes, in nontechnical language, a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health are identified by ATSDR and EPA.

Each profile includes the following:

(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects;

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure that present a significant risk to human health of acute, subacute, and chronic health effects; and

(C) Where appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

The principal audiences for the toxicological profiles are health professionals at the Federal, State, and local levels; interested private sector organizations and groups; and members of the public. We plan to revise these documents in response to public comments and as additional data become available. Therefore, we encourage comments that will make the toxicological profile series of the greatest use.

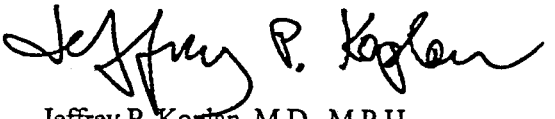
Comments should be sent to:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, N.E.
Mail Stop E-29
Atlanta, Georgia 30333

Background Information

The toxicological profiles are developed by ATSDR pursuant to Section 104(i) (3) and (5) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund) for hazardous substances found at Department of Energy (DOE) waste sites. CERCLA directs ATSDR to prepare toxicological profiles for hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL) and that pose the most significant potential threat to human health, as determined by ATSDR and the EPA. ATSDR and DOE entered into a Memorandum of Understanding on November 4, 1992 which provided that ATSDR would prepare toxicological profiles for hazardous substances based upon ATSDR's or DOE's identification of need. The current ATSDR priority list of hazardous substances at DOE NPL sites was announced in the Federal Register on July 24, 1996 (61 FR 38451).

This profile reflects ATSDR's assessment of all relevant toxicologic testing and information that has been peer-reviewed. Staff of the Centers for Disease Control and Prevention and other Federal scientists have also reviewed the profile. In addition, this profile has been peer-reviewed by a nongovernmental panel and is being made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.



Jeffrey P. Koplan, M.D., M.P.H.
Administrator
Agency for Toxic Substances and
Disease Registry

QUICK REFERENCE FOR HEALTH CARE PROVIDERS

Toxicological Profiles are a unique compilation of toxicological information on a given hazardous substance. Each profile reflects a comprehensive and extensive evaluation, summary, and interpretation of available toxicologic and epidemiologic information on a substance. Health care providers treating patients potentially exposed to hazardous substances will find the following information helpful for fast answers to often-asked questions.

Primary Chapters/Sections of Interest

Chapter 1: Public Health Statement: The Public Health Statement can be a useful tool for educating patients about possible exposure to a hazardous substance. It explains a substance's relevant toxicologic properties in a nontechnical, question-and-answer format, and it includes a review of the general health effects observed following exposure.

Chapter 2: Relevance to Public Health: The Relevance to Public Health Section evaluates, interprets, and assesses the signification of toxicity data to human health.

> ***NOTE:*** Not all health effects reported in this section are necessarily observed in the clinical setting. Please refer to the Public Health Statement to identify general health effects observed following exposure.

Pediatrics: Four new sections have been added to each Toxicological Profile to address child health issues:

Section 1.7	**How Can (Chemical X) Affect Children?**
Section 1.8	**How Can Families Reduce the Risk of Exposure to (Chemical X)?**
Section 3.6	**Children's Susceptibility**
Section 6.6	**Exposures of Children**

Other Sections of Interest:

Section 3.7	**Biomarkers of Exposure and Effect**
Section 3.10	**Methods for Reducing Toxic Effects**

ATSDR Information Center

Phone: 1-888-42-ATSDR or (404) 498-0110 ***Fax:*** (404) 498-0057
E-mail: atsdric@cdc.gov ***Internet:*** http://www.atsdr.cdc.gov

The following additional material can be ordered through the ATSDR Information Center:

Case Studies in Environmental Medicine: Taking an Exposure History—The importance of taking an exposure history and how to conduct one are described, and an example of a thorough exposure history is provided. Other case studies of interest include *Reproductive and Developmental Hazards; Skin Lesions and Environmental Exposures; Cholinesterase-Inhibiting Pesticide Toxicity;* and numerous chemical-specific case studies.

Managing Hazardous Materials Incidents is a three-volume set of recommendations for on-scene (prehospital) and hospital medical management of patients exposed during a hazardous materials incident. Volumes I and II are planning guides to assist first responders and hospital emergency department personnel in planning for incidents that involve hazardous materials. Volume III—*Medical Management Guidelines for Acute Chemical Exposures*—is a guide for health care professionals treating patients exposed to hazardous materials.

Fact Sheets (ToxFAQs) provide answers to frequently asked questions about toxic substances.

Other Agencies and Organizations

The National Center for Environmental Health (NCEH) focuses on preventing or controlling disease, injury, and disability related to the interactions between people and their environment outside the workplace. *Contact:* NCEH, Mailstop F-29, 4770 Buford Highway, NE, Atlanta, GA 30341-3724 • Phone: 770-488-7000 • FAX: 770-488-7015.

The National Institute for Occupational Safety and Health (NIOSH) conducts research on occupational diseases and injuries, responds to requests for assistance by investigating problems of health and safety in the workplace, recommends standards to the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA), and trains professionals in occupational safety and health. *Contact:* NIOSH, 200 Independence Avenue, SW, Washington, DC 20201 • Phone: 800-356-4674 or NIOSH Technical Information Branch, Robert A. Taft Laboratory, Mailstop C-19, 4676 Columbia Parkway, Cincinnati, OH 45226-1998 • Phone: 800-35-NIOSH.

*The National Institute of Environmental Health Sciences (*NIEHS) is the principal federal agency for biomedical research on the effects of chemical, physical, and biologic environmental agents on human health and well-being. *Contact:* NIEHS, PO Box 12233, 104 T.W. Alexander Drive, Research Triangle Park, NC 27709 • Phone: 919-541-3212.

Referrals

The Association of Occupational and Environmental Clinics (AOEC) has developed a network of clinics in the United States to provide expertise in occupational and environmental issues. *Contact:* AOEC, 1010 Vermont Avenue, NW, #513, Washington, DC 20005 • Phone: 202-347-4976 • FAX: 202-347-4950 • e-mail: aoec@dgs.dgsys.com • AOEC Clinic Director: http://occ-env-med.mc.duke.edu/oem/aoec.htm.

The American College of Occupational and Environmental Medicine (ACOEM) is an association of physicians and other health care providers specializing in the field of occupational and environmental medicine. *Contact:* ACOEM, 55 West Seegers Road, Arlington Heights, IL 60005 • Phone: 847-818-1800 • FAX: 847-818-9266.

CONTRIBUTORS

CHEMICAL MANAGER(S)/AUTHORS(S):

Malcolm Williams, Ph.D.
Sam Keith, M.S., C.H.P
John Risher, Ph.D.
ATSDR, Division of Toxicology, Atlanta, GA

David W Wohlers, Ph.D.
Mario Citra, Ph.D.
Gary L. Diamond, Ph.D.
Syracuse Research Corporation, North Syracuse, NY

Stephen G. Swarts, Ph.D.
Wake Forest University, Winston-Salem, NC

THE PROFILE HAS UNDERGONE THE FOLLOWING ATSDR INTERNAL REVIEWS:

1. Health Effects Review. The Health Effects Review Committee examines the health effects chapter of each profile for consistency and accuracy in interpreting health effects and classifying end points.

2. Minimal Risk Level Review. The Minimal Risk Level Workgroup considers issues relevant to substance-specific minimal risk levels (MRLs), reviews the health effects database of each profile, and makes recommendations for derivation of MRLs.

3. Data Needs Review. The Research Implementation Branch reviews data needs sections to assure consistency across profiles and adherence to instructions in the Guidance.

PEER REVIEW

A peer review panel was assembled for cesium. The panel consisted of the following members:

1. Herman Cember, C.H.P., Ph.D., P.E., Adjunct Professor, School of Health Sciences, Purdue University, Lafayette, Indiana;

2. Darrell Fischer, Ph.D., Senior Scientist, Pacific Northwest National Laboratory, Richland, Washington;

3. Marvin Goldman, Ph.D., Professor Emeritus of Radiation Biology, Department of Surgical and Radiological Sciences, University of California, Davis, California;

4. Bruce Muggenburg, D.V.M., Ph.D., Senior Scientist and Veterinary Physiologist, Toxicology Division, Lovelace Respiratory Research Institute, Albuquerque, New Mexico.

These experts collectively have knowledge of cesium's physical and chemical properties, toxicokinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in Section 104(I)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers' comments and determined which comments will be included in the profile. A listing of the peer reviewers' comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.

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1. PUBLIC HEALTH STATEMENT

This public health statement tells you about cesium and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Stable cesium has been found in at least 10 of the 1,585 current or former NPL sites. It was reported that ^{134}Cs has been found in at least 3 of the 1,585 current or former NPL sites and ^{137}Cs has been detected in at least 22 of the 1,585 current or former NPL sites. However, the total number of NPL sites evaluated for this substance is not known. As more sites are evaluated, the sites at which cesium is found may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance, or by skin contact.

If you are exposed to cesium, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT IS CESIUM?

Cesium is a naturally occurring element found in rocks, soil, and dust at low concentrations. Granites contain an average cesium concentration of about 1 part of cesium in a million parts of granite (ppm) and sedimentary rocks contain about 4 ppm. Natural cesium is present in the environment in only one stable form (isotope), ^{133}Cs. Pure cesium metal is silvery white in color and very soft, but pure cesium is not expected to be found in the environment. Pure cesium

metal reacts violently with air and water resulting in an explosion-like reaction. Cesium compounds do not react violently with air or water and are generally very soluble in water. The most important source of commercial cesium is a mineral known as pollucite, which usually contains about 5–32% cesium oxide (Cs_2O). No known taste or odor is associated with cesium compounds. Cesium is not mined or produced in the United States and very little is imported from other countries. There are relatively few commercial uses for cesium metal and its compounds. Sometimes cesium is used to absorb for residual gas impurities in vacuum tubes and as a coating in tungsten filaments or cathodes of the tubes. Cesium iodide and cesium fluoride are used in scintillation counters, which convert energy from ionizing radiation into pulses of visible light. Cesium is also used in highly accurate atomic clocks. For more information on the physical and chemical properties and on the production and use of cesium, see Chapters 4 and 5.

Radioactive forms of cesium are produced by the fission of uranium in fuel elements (fuel rods) during the normal operation of nuclear power plants, or when nuclear weapons are exploded. Radioactive forms of cesium are unstable and eventually change into other more stable elements through the process of radioactive decay. The two most important radioactive isotopes of cesium are ^{134}Cs and ^{137}Cs. Radioactive isotopes are constantly changing into different isotopes by giving off radiation. Each atom of ^{134}Cs changes into either xenon-134 (^{134}Xe) or barium-134 (^{134}Ba) neither of which is radioactive, while each atom of ^{137}Cs decays to barium-137 (^{137}Ba), which is also not radioactive. As ^{134}Cs and ^{137}Cs decay, beta particles and gamma radiation are given off. The half-life is the time it takes for half of that cesium isotope to give off its radiation and change into a different element. The half-life of ^{134}Cs is about 2 years and the half-life of ^{137}Cs is about 30 years.

1.2 HOW DO WE MEASURE RADIOACTIVITY?

Cesium is measured in units of mass (grams) or radioactivity (curies or becquerels). Both the curie (Ci) and the becquerel (Bq) tell us how much a radioactive material decays every second. The Bq is a new international unit known as the SI unit, and the Ci is an older unit, but it is more commonly used. The Ci was the original unit used to describe the intensity of radioactivity.

One Ci is equal to 37 billion radioactive emissions per second; this is approximately the radioactivity of 1 gram of radium. One Bq is equal to 1 radioactive emission per second. Thus,

1 Ci=37,000,000,000 Bq and

1 Bq=0.000000000027 Ci.

The table below gives the conversion between these units and the prefixes used. You can use the table to convert from one unit to the other. To do this, look at the first row of the table. You will see that 1 kilocurie (1 kCi) is equal to 37 terabequerels (37 TBq). To convert a different amount of radioactivity, say 5 pCi to Bq, using the table, you would first find that 1 pCi is equal to 37 mBq. Then you would multiply the 37 mBq by 5 to obtain 185 mBq (5 pCi=185 mBq).

Conversion of Radioactivity Units

Conventional Units			SI Units		
	prefix			prefix	
1 kCi	kilo	$1x10^{3}$ Ci	37 TBq	tera	$37x10^{12}$ Bq
1 Ci	—	$1x10^{0}$ Ci	37 GBq	giga	$37x10^{9}$ Bq
1 mCi	milli	$1x10^{-3}$ Ci	37 MBq	mega	$37x10^{6}$ Bq
1 µCi	micro	$1x10^{-6}$ Ci	37 kBq	kilo	$37x10^{3}$ Bq
1 nCi	nano	$1x10^{-9}$ Ci	37 Bq	—	$37x10^{0}$ Bq
1 pCi	pico	$1x10^{-12}$ Ci	37 mBq	milli	$37x10^{-3}$ Bq
1 fCi	femto	$1x10^{-15}$ Ci	37 µBq	micro	$37x10^{-6}$ Bq
1 aCi	atto	$1x10^{-18}$ Ci	37 nBq	nano	$37x10^{-9}$ Bq

Conversion of Radioactivity Units

SI Units			Conventional Units		
	prefix			prefix	
1 TBq	tera	$1x10^{12}$ Bq	27 Ci	—	$27x10^{0}$ Ci
1 GBq	giga	$1x10^{9}$ Bq	27 mCi	milli	$27x10^{-3}$ Ci
1 MBq	mega	$1x10^{6}$ Bq	27 µCi	micro	$27x10^{-6}$ Ci
1 kBq	kilo	$1x10^{3}$ Bq	27 nCi	nano	$27x10^{-9}$ Ci
1 Bq	—	$1x10^{0}$ Bq	27 pCi	pico	$27x10^{-12}$ Ci
1 mBq	milli	$1x10^{-3}$ Bq	27 fCi	femto	$27x10^{-15}$ Ci

1.3 WHAT HAPPENS TO CESIUM WHEN IT ENTERS THE ENVIRONMENT?

Naturally occurring cesium occurs in the environment mostly from the erosion and weathering of rocks and minerals. The mining and milling of certain ores can also release cesium to the air, water, and soil. Radioactive cesium is released to the environment during the normal operation of nuclear power plants, explosion of nuclear weapons, and accidents involving nuclear power plants or nuclear powered satellites or submarines.

Non-radioactive cesium can be neither created nor destroyed under typical environmental conditions, but can react with other compounds found in the environment and change into different cesium compounds. Radioactive decay is the only way for decreasing the concentration of ^{134}Cs and ^{137}Cs. Both stable and radioactive cesium are the same element and behave in a similar manner chemically and in the body. Cesium can travel long distances in the air before being brought back to the earth by rainfall and gravitational settling. In water and moist soils, most cesium compounds are very soluble. Cesium binds strongly to most soils and does not travel far below the surface of the soil. Consequently, it is not readily available for uptake by vegetation through roots. However, radiocesium can enter plants that fall onto the surface of leaves.

1.4 HOW MIGHT I BE EXPOSED TO CESIUM?

You can be exposed to stable or radioactive cesium by breathing air, drinking water, or eating food containing cesium. The level of cesium in air and water is generally low. The concentration of natural cesium in air is generally less than 1 nanogram (1 nanogram equals 1/1,000,000,000 of a gram) per cubic meter of air (ng/m^3). Cesium concentrations in drinking water is ordinarily about 1 microgram (1 microgram equals 1/1,000,000 of a gram) per liter of water (µg/L). On average, a person swallows about 10 µg of stable cesium per day in food and water, and breathes about 0.025 µg per day. Plants and animals have been shown to contain cesium at concentrations of about 1–300 ng/g.

Radioactive cesium has been detected at some level in surface water and in many types of food. This includes breast milk and pasteurized milk. The amount of radioactive cesium in food and milk is highly dependent upon several factors. The most important factor is whether or not there has been recent fallout from a nuclear explosion such as a weapons test or an accident that has occurred at a nuclear power plant. However, atmospheric testing of nuclear weapons was halted many years ago, and there have only been two major accidents at nuclear plants where radiocesium was released in significant amounts. The two accidents occurred in Windscale, England in 1957 and Chernobyl, Russia in 1986. You should understand that cesium only contributed a small fraction of the total radioactivity released following these events. Furthermore, the consequences of external exposure to gamma radiation and beta particles are not unique to ^{137}Cs and ^{134}Cs, but are very similar for all gamma and beta emitting radionuclides. People who work in industries that process or use natural cesium or cesium compounds can be exposed to higher-than-normal levels of cesium. An estimated 16,461 workers (4,276 of these are female) are potentially exposed to natural cesium and cesium compounds in the United States. If you work in the nuclear power industry, you may also be exposed to high levels of radioactive cesium, but there are many precautionary measures taken at these facilities to minimize this exposure.

1.5 HOW CAN CESIUM ENTER AND LEAVE MY BODY?

Stable and radioactive cesium can enter your body from the food you eat or the water you drink, from the air you breathe, or from contact with your skin. When you eat, drink, breathe, or touch things containing cesium compounds that can easily be dissolved in water, cesium enters your blood and is carried to all parts of your body. Cesium is like potassium; it enters cells and helps to maintain a balance of electrical charges between the inside and the outside of cells so that cells can perform tasks that depend on those electrical charges. Cells like muscle cells and nerve cells require changing electrical charges in order to function properly and allow you to think and move.

Once cesium enters your body, your kidneys begin to remove it from the blood; some cesium is quickly released from your body in the urine. A small portion is also released in the feces.

Some of the cesium that your body absorbs can remain in your body for weeks or months, but is slowly eliminated from your body through the urine and feces.

1.6 HOW CAN CESIUM AFFECT MY HEALTH?

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body. In the case of a radioactive chemical, it is also important to gather information concerning the radiation dose and dose rate to the body. For some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

You are not likely to experience any health effects that could be related to stable cesium itself. Animals given very large doses of cesium compounds have shown changes in behavior, such as increased activity or decreased activity, but it is highly unlikely that you would breathe, eat, or drink amounts of stable cesium large enough to cause similar effects. If you were to breathe, eat, drink, touch, or come close to large amounts of radioactive cesium, cells in your body could become damaged from the radiation that might penetrate your entire body, much like x-rays, even if you did not touch the radioactive cesium. You might also experience acute radiation syndrome, which includes such effects as nausea, vomiting, diarrhea, bleeding, coma, and even death. A number of people in Brazil, who played with radioactive cesium that was stolen from a medical machine used for radiation therapy, became sick from exposure to the radiation; a few of them died. But people exposed to radioactive cesium that has been widely dispersed in air, water, soil, or food following nuclear bombings or accidents have not been exposed to amounts large enough to cause the same effects.

1.7 HOW CAN CESIUM AFFECT CHILDREN?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans.

Children can be affected by cesium in the same ways as adults. Cesium is not likely to affect the health of children, but large amounts of gamma radiation, from sources such as radioactive cesium, could damage cells and might also cause cancer. Short exposure to extremely large amounts of radiation might cause nausea, vomiting, diarrhea, bleeding, coma, and even death. In addition, if babies were to be exposed to enough radiation while in their mother's womb during the time when their nervous system is rapidly developing, they could experience changes in their brains that could result in changes in behavior or decreased mental abilities. However, it is unlikely that children or babies would be exposed to enough gamma radiation from a radioactive cesium source to do such damage to their bodies.

1.8 HOW CAN FAMILIES REDUCE THE RISK OF EXPOSURE TO CESIUM?

If your doctor finds that you have been exposed to significant amounts of cesium, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate.

Stable Cesium. Since cesium is naturally found in the environment, we cannot avoid being exposed to it. However, the relatively low concentrations present do not warrant any immediate steps to reduce exposure.

Radioactive Cesium. You are unlikely to be exposed to high levels of radioactive cesium unless there is a fuel meltdown and accidental release at a nuclear power plant or a nuclear weapon has been detonated. In such cases, follow the advice of public health officials who will publish guidelines for reducing exposure to radioactive material when necessary.

1.9 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO CESIUM?

Everyone has small amounts of cesium in their body. Laboratories use special techniques to measure the amount of cesium in body fluids such as blood and urine, as well as in feces or other human samples. This can give an indication of whether a person has been exposed to levels of cesium that are higher than those normally found in food, water, or air. Special radiation detectors can be used to detect if a person has absorbed radioactive cesium. It is difficult to determine if a person has been exposed only to external radiation from radioactive cesium. Health professionals examining people who have health problems similar to those resulting from radiation exposure would need to rely on additional information in order to establish if such people had been near a source of radioactivity.

1.10 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), the Food and Drug Administration (FDA), and the Nuclear Regulatory Commission (NRC).

Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH), and the FDA.

Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals; then they are adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of

different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it. Some regulations and recommendations for cesium include the following:

There are few guidelines for compounds of stable cesium. Based on eye irritation, the NIOSH has established a recommended exposure limit (REL) for cesium hydroxide of 2 mg/m^3 as a time-weighted average (TWA) for up to a 10-hour workday and a 40-hour workweek. The American Conference of Governmental Industrial Hygienists (ACGIH) has assigned cesium hydroxide a threshold limit value (TLV) of 2 mg/m^3 as a TWA for a normal 8-hour workday and a 40-hour workweek, based on respiratory and eye irritation.

Radioactive cesium. The NRC established occupational inhalation exposure derived air concentrations (DACs) of 0.00000004 μCi/mL ($4x10^{-8}$ μCi/mL) for ^{134}Cs and 0.00000006 μCi/mL ($6x10^{-8}$ μCi/mL) for ^{137}Cs; annual limit intakes (ALIs) for on-the-job exposure are 100 μCi ($1x10^{2}$ μCi) for ^{134}Cs and 200 μCi ($2x10^{2}$ μCi) for ^{137}Cs.

The NRC also established limits for effluent concentrations of 0.0000009 μCi/mL ($9x10^{-7}$ μCi/mL) for ^{134}Cs and 0.000001 μCi/mL ($1x10^{-6}$ μCi/mL) for ^{137}Cs in water, as well as 0.0000000002 μCi/mL ($2x10^{-10}$ μCi/mL) for both ^{134}Cs and ^{137}Cs in air.

More information on regulations and guidelines is available in Chapter 8.

1.11 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department, your regional Nuclear Regulatory Commission office, or

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop E-29
Atlanta, GA 30333

* Information line and technical assistance

Phone: 1-888-42-ATSDR (1-888-422-8737)
Fax: (404) 498-0057

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

* To order toxicological profiles, contact

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161
Phone: 1-800-553-6847 or (703) 605-6000

2. RELEVANCE TO PUBLIC HEALTH

2.1 BACKGROUND AND ENVIRONMENTAL EXPOSURES TO CESIUM IN THE UNITED STATES

Naturally-occurring cesium exists as the stable isotope (^{133}Cs) in the earth's crust at an average concentration of about 1 ppm in granites and 4 ppm in sedimentary rocks. The most important source of commercial cesium is the mineral pollucite, which usually contains about 5–32% Cs_2O. The largest deposits of pollucite are located in Manitoba, Canada and account for about two-thirds of the world's known supply. Cesium has very low mobility in soil surfaces. Clay minerals and soils rich in exchangeable potassium adsorb cesium by binding the cations to interlayer positions of the clay particles. The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays, which can result in limited uptake of cesium by grass and plant material. Continental dust and soil erosion are the main emission sources of naturally-occurring cesium that is present in the environment. As a result of human activities, however, cesium is released into the environment globally in small amounts. Cesium has also been detected in the fly ash of hazardous waste incinerators and coal burning power plants. Cesium is deposited on plants and trees by wet and dry deposition and can be absorbed into the flora through its foliage. The deposited cesium can make its way to soil through litter decomposition. Due to its low relative abundance, limited use in industry, and relatively low level of toxicity observed in animal studies, exposure to stable cesium is not considered to be a significant public health concern.

Radioactive isotopes of cesium (^{134}Cs and ^{137}Cs) are formed during nuclear fission, being normally contained in commercial applications such as the generation of electricity at nuclear power plants. However, high levels of ^{134}Cs and ^{137}Cs have been released to the environment, as a result of atmospheric nuclear weapons testing (which has been discontinued for many years) and the accident at the Chernobyl nuclear reactor site in 1986. Once released, these radioactive cesium isotopes persist in the environment, posing potentially for adverse health effects. Following release to the atmosphere, radioactive cesium can travel thousands of miles before settling to earth, and is removed by wet and dry deposition. Radioactive cesium can also be released to soil or water in liquid effluents from spent fuel and fuel reprocessing plants.

The total amount of ^{137}Cs released from weapons testing through 1980 was estimated as $2.6x10^{7}$ Ci ($9.6x10^{17}$ Bq), 76% of which was released in the northern hemisphere and 24% in the southern hemisphere.

The accident at the Chernobyl nuclear power plant resulted in the release of an estimated $5.4x10^{5}$ Ci ($2.0x10^{16}$ Bq) of ^{134}Cs and $1.1x10^{6}$ Ci ($4.0x10^{16}$ Bq) of ^{137}Cs into the atmosphere over Europe. Routine activities at nuclear power plants and fuel-reprocessing stations also release ^{137}Cs and ^{134}Cs to the environment on a regular basis, but these are at such levels as to be considered insignificant. For example, $1.3x10^{-4}$ Ci ($4.8x10^{6}$ Bq) of ^{134}Cs and $5.1x10^{-3}$ Ci ($1.9x10^{8}$ Bq) of ^{137}Cs were released to the atmosphere in 1988, from the Savannah River plutonium processing site in South Carolina. In 1993, the Nuclear Regulatory Commission (NRC) estimated that 0.013 Ci ($4.8x10^{8}$ Bq) of ^{134}Cs and 0.023 Ci ($8.5x10^{8}$ Bq) of ^{137}Cs were released in airborne effluents from 30 PWR nuclear power plants operating in the United States. It was also estimated that $4.6x10^{-4}$ Ci ($1.7x10^{7}$ Bq) of ^{134}Cs and $3.3x10^{-3}$ Ci ($1.2x10^{8}$ Bq) of ^{137}Cs were released in airborne effluents from 28 BWR nuclear power plants.

During the period of 1961–1973, it was estimated that about 514 Ci of ^{137}Cs was emitted to the Savannah River watershed due to the activities at the Savannah River plutonium processing plant. In 1993, the NRC estimated that 1.88 Ci ($6.96x10^{10}$ Bq) of ^{134}Cs and 2.85 Ci ($1.05x10^{11}$ Bq) of ^{137}Cs were released in liquid effluents from 30 PWR nuclear power plants operating in the United States. The NRC estimated 0.12 Ci ($4.44x10^{9}$ Bq) of ^{134}Cs and 0.58 Ci ($2.15x10^{10}$ Bq) of ^{137}Cs were released in liquid effluents from 28 BWR nuclear power plants.

Since the half-life for some radioactive isotopes of cesium is long (the half-life of ^{137}Cs is about 30 years and the half-life of ^{134}Cs is about 2 years), the general population is exposed to ^{137}Cs and ^{134}Cs for long periods of time after it is released from a nuclear accident or weapons test, with the greatest exposure occurring near the source. Although inhalation and dermal exposure is possible, oral ingestion of contaminated food items is the greatest source of internal exposure for both naturally occurring and radioactive cesium. Workers employed in the mining and milling of pollucite ores and the production of cesium compounds are exposed to cesium through oral, dermal, and inhalation routes. Similar routes of exposure to ^{137}Cs and ^{134}Cs occurs for workers employed in the nuclear industry. External exposure to beta and gamma radiation can also occur for workers employed in the nuclear industry as well as for the general population following an accidental release or weapons test.

As discussed in Appendix A, the average annual effective dose of ionizing radiation (including ^{134}Cs and ^{137}Cs) from anthropogenic sources to the U.S. population is very small in comparison to natural sources.

2.2 SUMMARY OF HEALTH EFFECTS

Energy released by the radioactive isotopes can result in significant damage to living cells. Both ^{134}Cs and ^{137}Cs emit beta particles and gamma rays, which may ionize molecules within cells penetrated by these emissions and result in tissue damage and disruption of cellular function. The most important exposure routes for radioisotopes of cesium are external exposure to the radiation released by the radioisotopes and ingestion of radioactive cesium-contaminated food sources. Inhalation and dermal exposure routes may also present a health hazard. It should be noted that there is nothing unique about the dangers of external exposure to ^{134}Cs and ^{137}Cs when compared to other gamma and beta emitting radionuclides.

Radiation absorbed doses are expressed in terms of the amount of energy absorbed per unit mass, in units called rad or Gray (Gy) (see Appendix D and ATSDR 1999 for a complete description of principles of ionizing radiation). Generally, acute radiation doses below 15 rad (0.15 Gy) do not result in observable adverse health effects. At doses in the range of 15–50 rad (0.15–0.5 Gy), subclinical responses such as chromosomal breaks and transient changes in formed elements of the blood may be seen in sensitive individuals. Symptoms of acute radiation syndrome are observed at radiation doses above 50 rad, characterized by transient hematopoietic manifestations, nausea and vomiting, and moderate leukopenia at doses near 100 rad (1 Gy), progressing through more serious hematopoietic symptoms, clinical signs, and gastrointestinal symptoms with increasing dose (100–800 rad or 1–8 Gy), and usually death in persons receiving total doses $1,000 rad (10 Gy). Other health effects from acute or continued exposure to ionizing radiation may include reproductive, developmental, and latent cancer effects.

Signs and symptoms of acute toxicity from external and internal exposure to high levels of radiation from ^{134}Cs or ^{137}Cs are typical of those observed in cases of high exposure to ionizing radiation in general. Depending on the radiation dose, symptoms may include those typical of acute radiation syndrome (vomiting, nausea, and diarrhea), skin lesions, neurological signs, chromosomal abnormalities, compromised immune function, and death.

Acute or repeated exposure of humans or animals to ionizing radiation (from radioisotopes of cesium or other radioactive elements) may result in reduced male fertility, abnormal neurological development

following exposure during critical stages of fetal development, and genotoxic effects such as increased frequencies of chromosomal aberrations, T-lymphocyte point mutations, dominant lethal mutations, and reciprocal translocations.

Due to the ionizing properties of radionuclides such as ^{134}Cs and ^{137}Cs, increased cancer risk would be expected among exposed individuals. However, studies of increased cancer risk specifically associated with exposure of humans to radioactive cesium isotopes were not located. The only documented report of health effects in humans exposed to radioactive cesium as the source of radiation occurred in 1987, following the Chernobyl reactor accident in the Ukraine. Long-term cancer studies on exposed individuals have not been completed to date.

Animal studies indicate increased risk of cancer following external or internal exposure to relatively high doses of radiation from ^{137}Cs sources. Increased lifetime risk of mammary tumors was noted in female rats acutely exposed to whole-body radiation. Intravenous injection of ^{137}Cs (as cesium chloride) in dogs resulted in long-term increased risk of all cancers combined in males, and all cancers combined (excluding mammary cancer) in females.

Immunological and Lymphoreticular Effects. Humans accidently exposed externally and internally (oral and dermal) to ^{137}Cs that resulted in estimated radiation absorbed doses of 100–700 rad (1–7 Gy) exhibited severe bone marrow depression. Similar effects were seen in dogs exposed to ^{137}Cs by intravenous injection, resulting in estimated bone marrow doses of 700–2,400 rad (7–24 Gy).

Reproductive Effects. Exposure to radioisotopes of cesium may result in reduced fertility in males, as evidenced by reduced concentrations of spermatozoa in men who had been exposed externally and internally (dermal and oral) to ^{137}Cs approximately 1 month prior to testing. Reduced fertility, including sterility, was reported in male mice exposed to ^{137}Cs either by total-body external radiation, which resulted in a total radiation dose of 300 rad (3 Gy) over a 19.5-day exposure period, or by single or repeated oral dosing, which resulted in estimated total testicular radiation doses of 300–385 rad (3–3.85 Gy), measured at 5 weeks post-treatment. No significant reduction in male fertility was seen from total testicular radiation doses in the range of 10–100 rad (0.1–1 Gy). Persistent germinal epithelium damage and azoospermia were reported in all long-term surviving dogs that had been administered ^{137}Cs (as cesium chloride) by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 742 to 1,640 rad (7.42–16.40 Gy).

Developmental Effects. Developmental effects such as reduced post-natal body weight, impaired motor activity, morphological changes in the brain, reduced head size, and retarded odontogenesis and palatal closure have been reported in rats that had been exposed to radioactive cesium sources (^{137}Cs) *in utero* via whole-body external exposure of dams; effects were of largest magnitude when exposure occurred around gestational day 15. Reported developmental effects in similarly-exposed mice included significantly decreased brain weight and increased aggressive behavior. Atomic bomb survivors of Hiroshima and Nagasaki, exposed to high levels of ionizing radiation *in utero* during weeks 8–15 or 16–25 post-ovulation, exhibited later signs of impaired cognitive function. Radiation-induced developmental effects would be expected in humans or animals exposed to similar levels of ionizing radiation from any ionizing radiation source, including a radiocesium source. Resulting adverse health effects would be due to the radiation, not cesium *per se*.

Neurological Effects. Excess exposure to stable cesium appears to result in central nervous system effects. A man who voluntarily ingested cesium chloride daily for 36 days reported neurological signs that included feelings of euphoria, heightened sense perception, and tingling sensations within 15 minutes of dosing, in the absence of apparent adverse mental or motor skills. In animal studies, administration of cesium chloride has been reported to trigger stimulant and depressant central nervous system responses.

Since radioisotopes of cesium such as ^{134}Cs and ^{137}Cs emit beta particles and gamma rays capable of ionizing cells, acute radiation doses greater than 3,000 rad (30 Gy) would be expected to result in symptoms indicative of central nervous system syndrome that include immediate onset of violent nausea and vomiting, diarrhea, irrational behavior, circulatory system collapse, and neuromuscular incoordination, followed by convulsions, coma, and death within 48 hours (see ATSDR 1999 for more detailed information on health effects from exposure to ionizing radiation).

Dermal Effects. Human exposure to beta and gamma radiation from a radioactive cesium source, which had been stolen from a medical instrument, resulted in typical radiation-induced skin lesions. These lesions were due to the radiation, not to cesium *per se*.

Ocular Effects. Lacrimation, hyperemia and edema of the conjunctiva, and ocular pain were reported in some individuals following accidental external and internal (oral and dermal) exposure to beta and gamma radiation from a ^{137}Cs source. These lesions were due to the radiation, not to cesium *per se*.

Cancer. Studies that assess the risk of cesium-induced cancer are restricted to radioactive isotopes, not stable cesium. No human studies were located in which cancer incidence was specifically associated with exposure to radioisotopes of cesium. Due to the nature of ionizing radiation in general, carcinogenic effects similar to those observed in Japanese survivors of the 1945 atomic bombing incidents might be expected among individuals acutely exposed to high levels of radiation from a radioactive cesium source (see ATSDR 1999 for a detailed discussion of the carcinogenic effects of ionizing radiation). However, it is unlikely that levels of ionizing radiation as high as those experienced by the survivors of the atomic bombing incidents would be experienced by individuals who might be exposed to a radiocesium source. An exception is that of high-level exposure to a radiocesium source that had been removed from its protective shielding in Goiânia, Brazil, in 1987. Studies of long-term cancer risk are not yet available for this group of individuals.

Animal studies indicate increased risk of cancer following external or internal exposure to relatively high doses of radiation from ^{137}Cs sources. Increased lifetime risk of mammary tumors was noted in female rats acutely exposed to whole-body radiation. There were no significant differences between age groups irradiated at 8, 12, 16, 22, or 36 weeks of age, but irradiation at 64 weeks yielded fewer carcinomas than unirradiated controls. In lifetime studies of dogs administered single intravenous doses of ^{137}CsCl, which resulted in initial body burdens ranging from 1.0 to 4.0 mCi/kg (37–147 MBq/kg), benign and malignant neoplasms were found in a variety of tissues and organs, with no apparent single target organ of toxicity.

2.3 MINIMAL RISK LEVELS

Inhalation MRLs

No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for cesium due to the lack of suitable human or animal data regarding health effects following inhalation exposure to stable or radioactive cesium. Available information, considered relevant to inhalation exposure, is limited to two studies of dogs intravenously administered ^{137}CsCl. Adverse health effects included depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs. Striking similarities in the biokinetics, observed in laboratory animals exposed to ^{137}CsCl via either parenteral injection or inhalation or oral routes, indicate that adverse health effects might be common to all three routes of exposure. However, extrapolation of data across exposure routes was not considered to be a valid basis for the derivation of inhalation MRLs.

Oral MRLs

No acute-, intermediate-, or chronic-duration oral MRLs were derived for stable cesium due to the lack of suitable human or animal data regarding health effects following oral exposure to stable or radioactive cesium. The only available report regarding oral exposure to stable cesium in humans was that of an adult male daily ingesting approximately 68 mg Cs/kg (as cesium chloride) for 36 days; the man reported decreased appetite, nausea, and diarrhea, as well as neurological signs within 15 minutes following ingestion of the cesium chloride. Animal studies regarding oral exposure to stable cesium are limited to LD_{50} studies that indicate relatively low toxicity for stable cesium compounds. Information regarding human exposure to radioactive cesium is inadequate because no human data were available on health effects from oral exposure to radioactive cesium. Oral data regarding health effects in animals exposed to radioactive cesium are restricted to a single study in which only reproductive and genotoxic end points were reported.

Due to striking similarities in the biokinetics, observed in laboratory animals exposed to ^{137}CsCl via either parenteral injection or inhalation or oral routes, it has been suggested that adverse health effects might be common to all three routes of exposure. Depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs were observed in dogs intravenously administered ^{137}CsCl. However, extrapolation of data across exposure routes was not considered to be a valid basis for the derivation of oral MRLs.

MRLs for External Exposure to Cesium Isotopes

Two MRLs have been derived for ionizing radiation and are applicable to external exposure to radioisotopes of cesium:

- An MRL of 400 mrem (4.0 mSv) has been derived for acute-duration external exposure to ionizing radiation (14 days or less).

The acute MRL is based on results of a study by Schull et al. in which neurological effects of radiation, measured by intelligence test scores, were evaluated in children 10–11 years of age who had been exposed at critical stages of fetal development (gestation weeks 8–15) during the atomic bombing of Hiroshima and Nagasaki. When IQ scores were regressed on radiation dose estimates, IQ diminished linearly with increasing dose, resulting in an estimated decrease in IQ score of approximately 25 points

per 100 rad (100 rem in dose equivalent) or 0.25 points/rem (25 points/Sv). To derive the MRL of 400 mrem (4.0 mSv), ATSDR divided the dose associated with a predicted change of 0.25 IQ points (1 rem) by an uncertainty factor of 3 (for human variability/sensitive population). ATSDR noted that a change in IQ points of 0.25 is less than the reported difference of 0.3 IQ points between separated and unseparated identical twins.

The Nuclear Regulatory Commission (NRC) set a radiation exposure limit of 500 mrem (5 mSv) for pregnant working women over the full gestational period. For the critical gestational period of 8–15 weeks, ATSDR believes that the acute MRL of 400 mrem (4 mSv) is consistent with the NRC limit and could be applied to either acute (0–14 day) or intermediate (15–365 day) exposure periods.

- An MRL of 100 mrem/year (1.0 mSv/year) above background has been derived for chronic-duration external exposure to ionizing radiation (365 days or more).

The MRL is based on the BEIR V report that the average annual effective ionizing radiation dose to the U.S. population is 360 mrem/year (3.6 mSv/year), a dose not expected to produce adverse health effects. This dose is obtained mainly by naturally-occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. An uncertainty factor of 3 (for human variability) was applied to the NOAEL of 360 mrem/year to derive the MRL of 100 mrem/year.

3. HEALTH EFFECTS

3.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective on the toxicology of cesium, existing naturally as the stable (nonradioactive) isotope ^{133}Cs and in the form of radioactive isotopes produced during nuclear fission, the most abundant of which are ^{137}Cs and ^{134}Cs. This chapter contains descriptions and evaluations of toxicological studies and epidemiological investigations and provides conclusions, where possible, on the relevance of toxicity and toxicokinetic data to public health.

A glossary and list of acronyms, abbreviations, and symbols can be found at the end of this profile.

Health effects regarding the element cesium are categorized by the chemical nature of stable cesium (^{133}Cs) and the radioactive nature of unstable isotopes such as ^{137}Cs or ^{134}Cs, the chemical nature of internalized radioactive cesium being the same as that of stable cesium.

Since the average concentration of stable cesium in the earth's crust is low (on average about 1 ppm) and stable cesium is used only in small quantities in electronic and energy production industries, the risk of significant exposure to stable cesium via inhalation, oral, or dermal routes is expected to be small. Limited information is available on monitoring (or detection) of stable cesium in the environment and on health effects from exposure to stable cesium.

The only evidence of adverse effects in humans exposed to stable cesium is a report of decreased appetite, nausea, and diarrhea in a man who ingested about 68 mg Cs/kg/day (as cesium chloride) for 36 days; apparent neurological changes within 15 minutes of intake were also noted (Neulieb 1984).

Animal studies indicate that cesium is of relatively low toxicity. Acute oral LD_{50} values for rats and mice range from 800 to 2,000 mg Cs/kg, cesium hydroxide being more toxic than cesium iodide or cesium chloride. Single oral doses of cesium chloride, administered to female mice at dose levels ranging from 125 to 500 mg/kg, have been shown to result in significant increases in chromosomal breaks in bone marrow cells (Ghosh et al. 1990, 1991).

No reports were located regarding adverse effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation or dermal exposure to stable cesium.

Radioactive isotopes of cesium are a greater health concern than stable cesium. The most important exposure routes are external exposure to the radiation emitted by the radioisotope and ingestion of radioactive cesium-contaminated food sources. Vascular plants do not accumulate large levels of cesium through root uptake because cesium is strongly adsorbed to soils. However, the deposition of radioactive debris on flora with large surface areas such as lichens or moss is significant. Animals that feed on this vegetation, such as reindeer and caribou, may ingest large quantities of radiocesium (and other radionuclides found in fallout). Human consumption of meat from such animals results in the internalization of these radionuclides (see Section 6.7 for more detailed information on the lichen-caribou-human food chain). Radioactive cesium particles may be found in the air following the release of nuclear fission products; however, no reports were located in which adverse health effects from inhalation of radioactive cesium were discussed. Dermal absorption has been qualitatively described in rats, but no data were located on relative amounts and absorption rates (Pendic and Milivojevic 1966).

A number of individuals in Goiânia, Brazil, externally and internally (oral and dermal) exposed to a ^{137}Cs source, exhibited classic symptoms of acute radiation syndrome including vomiting, diarrhea, and nausea, as well as skin lesions from radiation burns, orofacial lesions, ocular injury, hematological effects (bone marrow aplasia, leukopenia, thrombocytopenia, lymphopenia, neutropenia), mild elevations of some liver enzymes, reduced sperm counts, and death (in four cases, attributed to infections resulting from reduced resistance) (Brandão-Mello et al. 1991; Gomes et al. 1990). External exposure was estimated based on frequencies of chromosomal aberrations in lymphocytes of exposed individuals at various times following exposure, while internal doses were estimated based on whole-body radiation counting and excretory levels of ^{137}Cs. The adverse effects were the result of beta and gamma radiation, not to cesium *per se*.

Although there is limited information regarding health effects in humans that can be exclusively associated with exposure to radioactive cesium sources, both ^{137}Cs and ^{134}Cs are products of nuclear fission and may, therefore, be released from sites where nuclear fission occurs, from radioactive material removed from such sites, or from leakage of radioactive cesium sources. These radionuclides (^{137}Cs and ^{134}Cs) emit beta particles (that travel short distances and can penetrate the skin and superficial body tissues) and gamma rays (that can penetrate the entire body). Gamma rays originating in radiocesium sources outside the body are the main source of external radiation hazard to internal organs. Once radioactive cesium is internalized, beta particles are the major cause of damage to nearby tissues. Tissue

damage results from the ionization of atoms that are encountered by ionizing (e.g., alpha, beta, gamma) radiation. In radiation biology, the term "dose" refers to the amount of energy (over time) imparted to tissue per gram of absorbing material. Absorbed dose is the amount of energy absorbed per unit mass, measured in units called rad or Gray (Gy) (see Appendix D for a detailed description of principles of ionizing radiation). Adverse health effects resulting from external exposure to beta or gamma emissions from radioisotopes of cesium would be the same as those from other radioactive elements that release beta or gamma radiation, and would not be the result of exposure to cesium *per se*. Developmental and carcinogenic effects have been reported in Japanese survivors of acute high-dose external radiation from the atomic bombs detonated over Hiroshima and Nagasaki (see ATSDR 1999 for a detailed description of the health effects related to ionizing radiation in general). Although such effects would be expected in individuals exposed to similar levels of external radiation from any source of gamma radiation, it is unlikely that such high levels would be achieved from any radiocesium source.

Information regarding adverse effects following inhalation, oral, or dermal exposure to radioactive isotopes of cesium was reported in two studies in which significantly reduced fertility and temporary sterility were observed in male mice following single or repeated oral administration of radioactive cesium nitrate (Ramaiya et al. 1994). Postmating embryo mortality was associated with increased frequency of dominant lethal mutations. No other studies were located regarding inhalation or oral exposure to radioactive cesium, since levels great enough to cause significant adverse health effects would also pose a severe health risk to investigators. However, it has been shown that distribution patterns of ^{137}Cs are similar in animals exposed to relatively nontoxic levels of ^{137}CsCl by parenteral injection, inhalation exposure, or oral administration (Boecker et al.1969a; Stara 1965). Therefore, the occurrence of depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues in dogs exposed to ^{137}CsCl via intravenous injection provide the most reasonable indication of health effects that would be expected in animals exposed to ^{137}CsCl by inhalation or oral exposure (Nikula et al. 1995, 1996).

Adverse neurological, developmental, reproductive, genotoxic, and cancer effects have been observed in animal studies employing external exposure to radioactive cesium sources, and are the result of the radiation, not the cesium *per se*. Impaired motor activity, decreased thickness of cortical layers of the brain, and increased aggressive behavior were observed after the birth of rats that had been briefly exposed *in utero* to relatively high levels of external radiation from a ^{137}Cs source (Minamisawa et al. 1992; Norton and Kimler 1987, 1988). The most vulnerable developmental period was around gestational days 14–15. In another study, adverse developmental effects in fetal rats irradiated on

gestational day 12 included reduced litter size, smaller head size, retarded odontogenesis, and cleft palate when examined on gestational day 18 (Saad et al. 1991, 1994). Significant increases in the formation rate of micronuclei were seen in blood cells of other fetal rats following irradiation of pregnant dams via a ^{137}Cs source on gestational day 14 (Koshimoto et al. 1994). Significantly reduced fertility (including temporary sterility) was reported in male mice exposed to an external ^{137}Cs source for almost 20 days; an increased frequency of dominant lethal mutations was also indicated by increased postmating embryo mortality (Ramaiya et al. 1994). Increased lifetime risk of mammary tumors was noted in female rats that were exposed, between the ages of 8 and 36 weeks, to single whole-body doses of radiation from a ^{137}Cs source (Bartstra et al. 1998). Irradiation at 64 weeks, however, yielded fewer carcinomas than unirradiated controls.

3.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals and others address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure (inhalation, oral, and dermal) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects). These data are discussed in terms of three exposure periods: acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies. LOAELS have been classified into "less serious" or "serious" effects. "Serious" effects are those that evoke failure in a biological system and can lead to morbidity or mortality (e.g., acute respiratory distress or death). "Less serious" effects are those that are not expected to cause significant dysfunction or death, or those whose significance to the organism is not entirely clear. ATSDR acknowledges that a considerable amount of judgment may be required in establishing whether an end point should be classified as a NOAEL, "less serious" LOAEL, or "serious" LOAEL, and that in some cases, there will be insufficient data to decide whether the effect is indicative of significant dysfunction. However, the Agency has established guidelines and policies that are used to classify these end points. ATSDR believes that there is sufficient merit in this approach to warrant an attempt at distinguishing between "less serious" and "serious" effects. The distinction between "less serious" effects and "serious" effects is considered to be important because it helps the users of the profiles to identify levels of exposure at which major health effects start to appear. LOAELs or NOAELs should also help in determining whether or not

the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the Levels of Significant Exposure (LSE) tables and figures may differ depending on the user's perspective. Public health officials and others concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAELs) or exposure levels below which no adverse effects (NOAELs) have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels or MRLs) may be of interest to health professionals and citizens alike.

Estimates of exposure levels posing minimal risk to humans (Minimal Risk Levels or MRLs) have been made for cesium. An MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure. MRLs are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration within a given route of exposure. MRLs are based on noncancerous health effects only and do not consider carcinogenic effects. MRLs can be derived for acute, intermediate, and chronic duration exposures for inhalation and oral routes. Appropriate methodology does not exist to develop MRLs for dermal exposure.

Although methods have been established to derive these levels (Barnes and Dourson 1988; EPA 1990b), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs will be revised.

A User's Guide has been provided at the end of this profile (see Appendix B). This guide should aid in the interpretation of the tables and figures for Levels of Significant Exposure and the MRLs.

3.2.1 Inhalation Exposure

During and after nuclear accidents, such as the steam explosion that occurred at the Chernobyl nuclear power plant in 1986, significant amounts of ^{137}Cs (and to a lesser extent ^{134}Cs) may be released to the atmosphere in suspended particles and widely dispersed through the air. Although radioactive cesium suspended in the air following such accidents or re-suspended later from ground-deposited fallout (NRC 1998) may be internalized via inhalation, there was no indication that inhalation was a significant route of exposure to radioactive cesium among individuals exposed externally by either being in the vicinity of a release or in areas receiving substantial ground-deposited fallout, or those exposed by ingestion of radioactive cesium-contaminated food following the Chernobyl accident (Balonov 1993).

No reports were located regarding health effects in humans or animals following inhalation exposure to cesium. Available human case reports and animal studies involving inhaled radioisotopes of cesium deal exclusively with biokinetics. Parenteral injection of ^{137}CsCl in laboratory animals has resulted in distribution patterns and tissue doses of ^{137}Cs that are similar to those resulting from inhalation or oral exposure (Boecker et al.1969a; Stara 1965). For these reasons, it has been proposed that adverse health effects, related to a soluble and readily absorbed compound such as ^{137}CsCl, should be similar across the three routes of exposure (Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Therefore, depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs, effects that have been observed in dogs following intravenous administration of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972), would be expected to occur following inhalation exposure to air concentrations of ^{137}CsCl that would result in comparable ^{137}Cs blood concentrations (see Section 3.2.5 for additional information regarding exposure other than inhalation, oral, dermal, or external exposure).

3.2.1.1 Death

No studies were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to stable cesium.

Although no studies were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium, dose-related decreased survival was observed in beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens 1.9–4.0 mCi/kg (71.7–147 MBq/kg) (Nikula et al. 1995, 1996). Similar

results would be expected in animals exposed to air concentrations of ^{137}CsCl that would result in similar body burdens (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

All reliable LOAEL values for death in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-1 and plotted in Figure 3-1, since the same effect would be expected to occur in dogs following inhalation exposure to ^{137}CsCl at levels that would result in comparable ^{137}Cs blood levels.

3.2.1.2 Systemic Effects

No data were located regarding systemic effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to stable or radioactive cesium. However, hematological effects similar to those observed in dogs that had received single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972) would be expected in animals exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection.

The highest NOAEL values and all reliable LOAEL values for systemic effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-1 and plotted in Figure 3-1, since the same effects would be expected to occur in dogs following inhalation exposure to ^{137}CsCl at levels that would result in comparable ^{137}Cs blood levels.

Hematological Effects. Depressed blood cell counts and platelet levels, reduced packed-cell volume, and bone marrow aplasia were observed in dogs that had been administered single intravenous injections of ^{137}CsCl, which resulted in average initial body burdens ranging from 1.0 to 3.8 mCi (36.4–141 MBq/kg) (Nikula et al. 1995; Redman et al. 1972). Severely depressed blood cell counts were observed in 23 dogs that died within 52 days following single intravenous administration of ^{137}CsCl at levels resulting in initial body burdens in the range of 1.7–4.4 mCi/kg (61–162 MBq/kg) (Nikula et al. 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

Table 3-1. Levels of Significant Exposure to Cesium - Chemical Toxicity - Oral

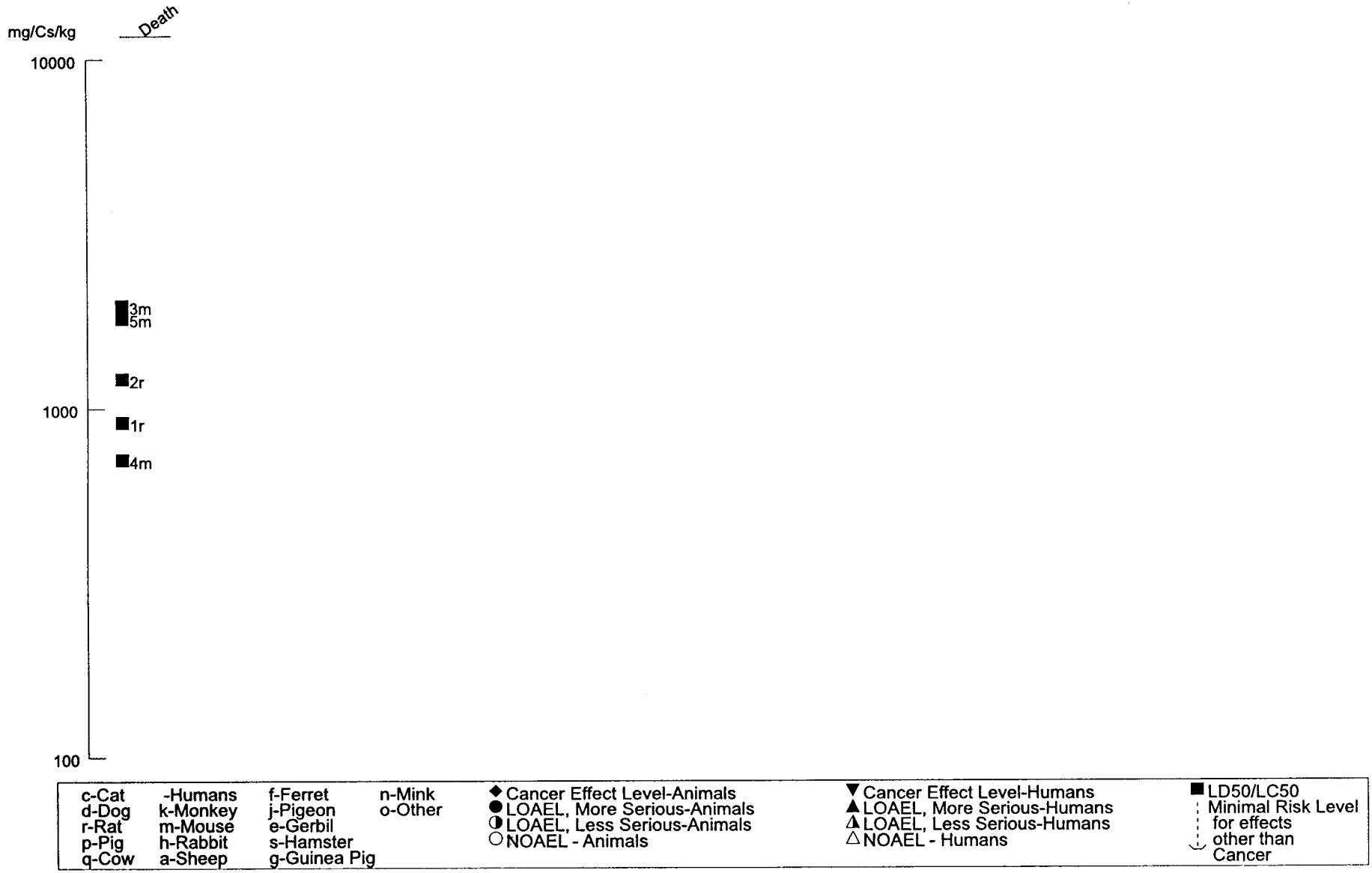
Key to[a] figure	Species (Strain)	Exposure/ duration/ frequency (Specific route)	System	NOAEL (mgCs/kg)	LOAEL Less serious (mgCs/kg)	LOAEL Serious (mgCs/kg)	Reference Chemical Form
	ACUTE EXPOSURE						
	Death						
1	Rat Charles River albino	Once (GW)				913 M (LD_{50})	Johnson et al. 1975 Cesium hydroxide
2	Rat Charles River albino	Once (GW)				1217 M (LD_{50})	Johnson et al. 1975 Cesium iodide
3	Mouse Swiss albino	Once (NS)				1975 F (LD_{50})	Ghosh et al. 1990 Cesium chloride
4	Mouse	Once				712 (LD_{50})	Khosid 1967 Cesium hydroxide
5	Mouse	Once				1817 (LD_{50})	Khosid 1967 Cesium chloride

[a]The number corresponds to entries in Figure 3-1.

F = female; (GW) = gavage in water; LD_{50} = lethal dose, 50% kill; LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; (NS) = not specified.

Figure 3-1. Levels of Significant Exposure to Cesium - Chemical Toxicity - oral

Acute (≤14 days)



3.2.1.3 Immunological and Lymphoreticular Effects

No data were located regarding immunological or lymphoreticular effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to stable or radioactive cesium. However, severe bone marrow depression was observed in dogs exposed to ^{137}Cs by intravenous injection at activity levels resulting in estimated total bone marrow doses of 700–2,400 rad (7–24 Gy) (Nikula et al. 1995). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

The highest NOAEL values and all reliable LOAEL values for immunological and lymphoreticular effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-1 and plotted in Figure 3-1, since the same effects would be expected to occur in dogs following inhalation exposure to ^{137}CsCl at levels that would result in comparable ^{137}Cs blood levels.

3.2.1.4 Neurological Effects

No data were located regarding neurological effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to stable or radioactive cesium.

3.2.1.5 Reproductive Effects

No data were located regarding reproductive effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to stable or radioactive cesium. However, germinal epithelium damage and azoospermia were reported in dogs that had been administered ^{137}Cs (as cesium chloride) by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 742 to 1,640 rad (7.42–16.40 Gy) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

The highest NOAEL values and all reliable LOAEL values for reproductive effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-1 and plotted in Figure 3-1, since the same effect

would be expected to occur in dogs following inhalation exposure to ^{137}CsCl at levels that would result in comparable ^{137}Cs blood levels.

3.2.1.6 Developmental Effects

No data were located regarding developmental effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to stable or radioactive cesium.

3.2.1.7 Cancer

No data were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration oral exposure to stable or radioactive cesium. However, benign and malignant neoplasms were found in a variety of tissues and organs of dogs administered single intravenous doses of ^{137}Cs (as cesium chloride), which resulted in initial body burdens ranging from 1.0 to 4.0 mCi/kg (37–147 MBq/kg) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

All Cancer Effect Level (CEL) values for dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-1 and plotted in Figure 3-1, since the same effect would be expected to occur in dogs following inhalation exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

3.2.2 Oral Exposure

No reports were located regarding health effects in humans or animals following oral exposure to potentially hazardous amounts of stable or radioactive cesium. Available human case reports and animal studies involving ingested radioisotopes of cesium deal exclusively with biokinetics. Parenteral injection of ^{137}CsCl in laboratory animals has resulted in distribution patterns and tissue doses of ^{137}Cs that are similar to those resulting from inhalation or oral exposure (Boecker et al.1969a; Stara 1965). For these reasons, it has been proposed that adverse health effects, related to a soluble and readily absorbed compound such as ^{137}CsCl, should be similar across the three routes of exposure (Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Therefore, depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of

tissues and organs, effects that have been observed in dogs following intravenous administration of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972), would be expected to occur following oral administration of ^{137}CsCl in amounts that would result in comparable ^{137}Cs blood concentrations (see Section 3.2.5 for additional information regarding exposure other than inhalation, oral, dermal, or external exposure).

3.2.2.1 Death

Stable Cesium. No reports were located regarding death in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No studies were located regarding death in animals following intermediate- or chronic-duration oral exposure to stable cesium. However, acute oral administration of cesium at high dose levels has resulted in observed mortality in rats and mice. In female mice administered cesium chloride, reported oral LD_{50} values range from 2,300 to 2,500 mg/kg (Ghosh et al. 1990; Khosid 1967). An acute oral LD_{50} value for cesium iodide is 2,386 mg/kg in rats (Johnson et al. 1975). Cesium hydroxide appears to be more highly toxic to rats than cesium chloride and cesium iodide, as evidenced by a lower LD_{50} value of 1,026 mg/kg (Johnson et al. 1975). Some of the toxic effects can be attributed to the strong alkaline nature of cesium hydroxide. Khosid (1967) estimated an acute oral LD_{50} value of 800 mg/kg for mice that were administered cesium hydroxide. No information was located regarding mortality in animals following oral administration of other compounds of stable cesium.

The highest NOAEL values and all reliable LOAEL values for death from acute-duration oral exposure to stable cesium are presented in Table 3-2 and plotted in Figure 3-2.

Radioactive Cesium. No reports were located regarding death in humans that could be exclusively associated with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. In an event involving mixed external, dermal, and oral exposure of adults and children to a ^{137}Cs source, significant short-term morbidity was followed in 50 patients and 4 deaths were reported within a few weeks among individuals with estimated radiation doses ranging from 450 to 600 rad (4.5–6 Gy) (Brandão-Mello et al. 1991).

Although no studies were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium, decreased survival was observed in young adult

Table 3-2. Levels of Significant Exposure to Cesium - Radiation Toxicity - Oral

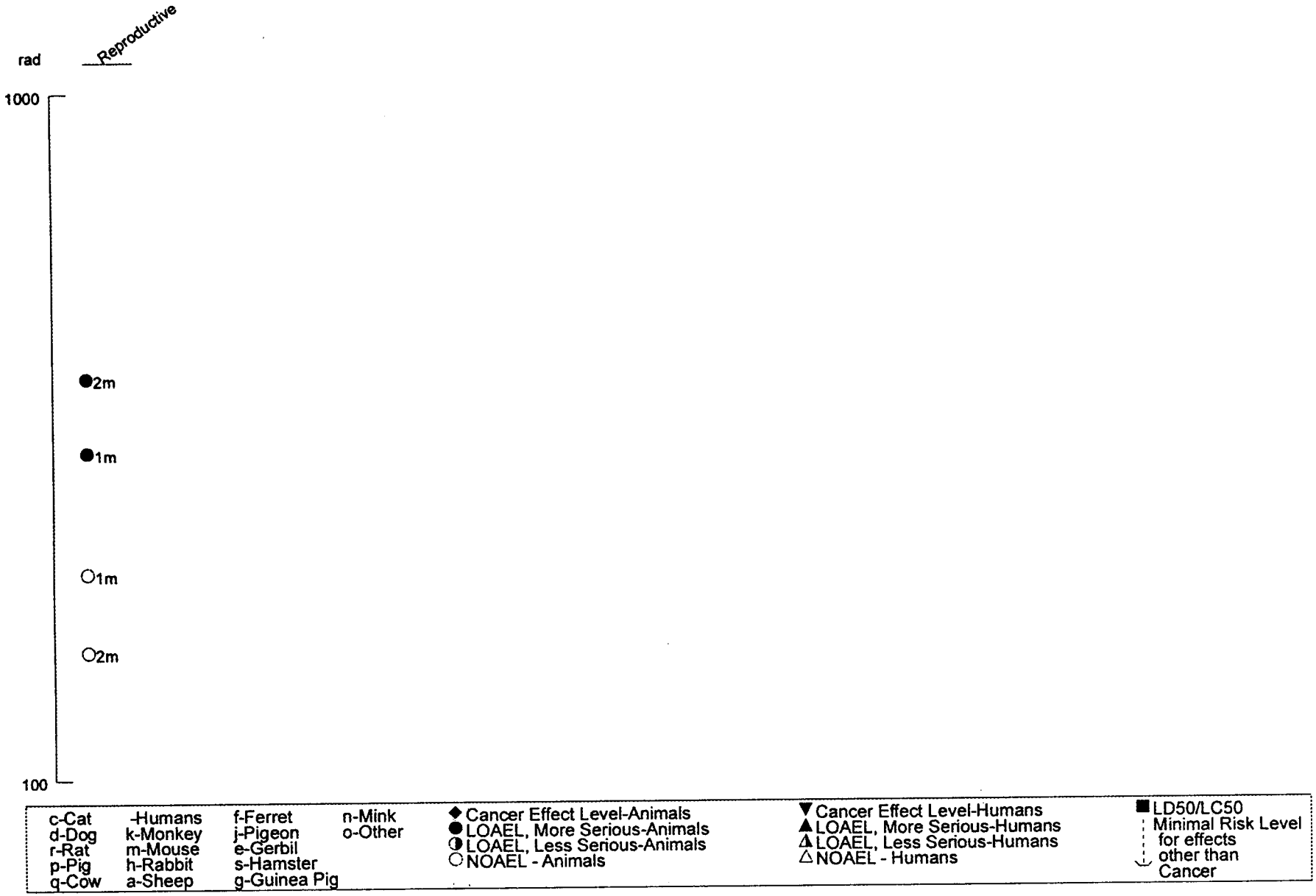
Key to figure[a]	Species (Strain)	Exposure/ duration/ frequency (Specific route)	System	NOAEL (rad)	LOAEL		Reference Chemical Form
					Less serious (rad)	Serious (rad)	
	ACUTE EXPOSURE						
	Reproductive						
1	Mouse (Hybrid)	Once (G)		200 M		300 M (temporary sterility)	Ramaiya et al. 1994 ^{137}Cesium nitrate
2	Mouse (Hybrid)	2 wk 1x/d (G)		154 M		385 M (reduced fertility)	Ramaiya et al. 1994 ^{137}Cesium nitrate

[a]The number corresponds to entries in Figure 3-2.

d = day(s); (G) = gavage; LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; wk = week(s).

Figure 3-2. Levels of Significant Exposure to Cesium - Radiation Toxicity - oral

Acute (≤14 days)



beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens of 1.9–4.0 mCi/kg (71.7–147 MBq/kg) (Nikula et al. 1995, 1996). Similar results would be expected in animals following oral exposure to ^{137}CsCl levels that would result in similar body burdens (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

All reliable LOAEL values for death in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-2 and plotted in Figure 3-2, since the same effect would be expected to occur in dogs following oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

3.2.2.2 Systemic Effects

Stable Cesium. Information regarding systemic effects in humans following oral exposure to stable cesium is restricted to a report of decreased appetite, nausea, and diarrhea in a man who ingested about 34 mg Cs/kg (as cesium chloride) after morning and evening meals for 36 days; this man also experienced apparent neurological changes within 15 minutes of dosing (Neulieb 1984).

No animal studies were located in which the systemic effects that were observed following oral administration of compounds of stable cesium could be attributed to the presence of cesium. Gastrointestinal and respiratory effects noted in rats administered acute oral doses of cesium hydroxide may have been due to the alkaline properties of the compound rather than the biochemical behavior of cesium.

Radioactive Cesium. Acute radiation syndrome, characterized by nausea, vomiting, and diarrhea was reported in a number of individuals exposed externally and internally (oral and dermal exposure) to a 1,375 Ci (50.9 TBq) ^{137}Cs source in Goiânia, Brazil. Other adverse effects included skin lesions, ocular lesions, severe bone marrow depression, and mild elevations in the activities of some liver enzymes (Brandão-Mello et al. 1991; Gomes et al. 1990; Rosenthal et al. 1991). Hematological effects similar to those observed in dogs that had received single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972) would be expected in animals following oral administration of ^{137}CsCl at levels that would result in body burdens similar to those attained via intravenous injection.

The highest NOAEL values and all reliable LOAEL values for systemic effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-2 and plotted in Figure 3-2, since the same

effects would be expected to occur in dogs following oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

No data were located regarding cardiovascular effects, musculoskeletal effects, endocrine effects, or metabolic effects in humans or animals following oral exposure to stable or radioactive cesium.

Respiratory Effects.

Stable Cesium. No reports were located regarding respiratory effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding respiratory effects in animals following intermediate- or chronic-duration exposure to stable cesium. Congested, cyanotic lungs with petechial hemorrhages were observed in rats following oral treatment with single cesium iodide doses large enough to cause death. A bloody nasal exudate was seen in some relatively high-dose rats (Johnson et al. 1975).

Radioactive Cesium. No reports were located for humans or animals that associate respiratory effects with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Gastrointestinal Effects.

Stable Cesium. No reports were located regarding gastrointestinal effects in humans following acute- or chronic-duration oral exposure to stable cesium, and only one report of intermediate-duration exposure was located. An investigator who voluntarily ingested about 34 mg Cs/kg (as cesium chloride, assuming 70 kg body weight) after each morning and evening meal (68 mg Cs/kg/day) for 36 days reported gradually decreased appetite, prenausea feelings, and diarrhea. The observations were self-described and effects were correlated to dietary habits during the course of the study (Neulieb 1984).

No reports were located regarding gastrointestinal effects in animals following intermediate- or chronic-duration oral exposure to stable cesium. In an acute lethality study of rats, administration of cesium hydroxide or cesium iodide at dose levels up to 910 or 1,217 mg Cs/kg, respectively, resulted in stomach and intestinal hemorrhage, bloody fluid exudate within the peritoneal cavity, and adhesions of abdominal organs. Rats receiving lethal doses of cesium iodide exhibited fluid-filled stomach (Johnson et al. 1975).

Radioactive Cesium. Vomiting, diarrhea, and nausea were observed among eight patients treated for acute radiation exposure to a ^{137}Cs source, both via external exposure and internal (oral and dermal) exposure (Brandão-Mello et al. 1991). These and other symptoms were classic symptoms of acute radiation syndrome. No reports were located regarding gastrointestinal effects in humans following intermediate- or chronic-duration oral exposure to radioactive cesium

No reports were located for animals regarding gastrointestinal effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Hematological Effects.

Stable Cesium. No reports were located regarding hematological effects in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

Radioactive Cesium. No reports were located for humans regarding hematological effects following intermediate- or chronic-duration oral exposure to radioactive cesium. In the 1987 incident of overexposure to a stolen medical radiation source containing 1,375 Ci (50.9 Tbq) ^{137}Cs, approximately 250 persons were contaminated externally, many of whom also were contaminated internally. Twenty individuals developed the acute radiation syndrome, 14 of whom developed bone marrow failure after having received whole-body radiation doses ranging from 100 to 700 rad (1.0–7.0 Gy). Four of these 14 heavily contaminated individuals died. These effects are typical signs and symptoms of the hemopoietic (blood forming) syndrome in which the blood forming cells in the bone marrow are killed. This results in sharp decreases of all the blood cells and consequent impairment of the immune system and anemia (Brandão-Mello et al. 1991; Gomes et al. 1990).

No reports were located for animals regarding hematological effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, depressed blood cell counts and platelet levels, reduced packed-cell volume, and bone marrow aplasia were observed in dogs that had been administered single intravenous injections of ^{137}CsCl, which resulted in average initial body burdens ranging from 1.0 to 3.8 mCi (36.4–141 Mbq/kg) (Nikula et al. 1995; Redman et al. 1972). Severely depressed blood cell counts were observed in 23 dogs that died within 52 days following single intravenous administration of ^{137}CsCl at levels resulting in initial body burdens in the range of 1.7–4.4 mCi/kg (61–162 MBq/kg) (Nikula et al. 1996). Similar effects would be expected in dogs following oral administration of ^{137}CsCl at levels that would result in body burdens similar to those

attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

Hepatic Effects.

Stable Cesium. No reports were located regarding hepatic effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding hepatic effects in animals following acute- or chronic-duration oral exposure to stable cesium. No significant effect on maternal liver weight was noted in rats consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

Radioactive Cesium. No reports were located that associate acute-duration oral exposure to radioactive cesium with hepatic effects. Mild elevations of aminotransferases (ALT/AST) were seen in a few patients hospitalized following radiation exposure to a ^{137}Cs source (Brandão-Mello et al. 1991). Exposures were most likely both external and internal (via oral and/or dermal routes). No reports were located for humans regarding hepatic effects following intermediate- or chronic-duration oral exposure to radioactive cesium.

No reports were located for animals regarding hepatic effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Renal Effects.

Stable Cesium. No reports were located regarding renal effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding renal effects in animals following acute- or chronic-duration oral intake of stable cesium, and one report was located regarding intermediate-duration intake. In that study, no significant effect on maternal kidney weight was noted in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

Radioactive Cesium. No reports were located for humans or animals that associate renal effects with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Dermal Effects.

Stable Cesium. No reports were located regarding dermal effects in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

Radioactive Cesium. Reports of dermal effects following external and internal (oral and dermal) exposure to radioactive cesium are restricted to the accidental exposure of a number of individuals to a ^{137}Cs source in which orofacial lesions, including oral bleeding and associated oral rash, mouth ulcers, acute oral candidiasis, and radiation dermatitis and depigmentation, were observed in 21 patients who had been acutely exposed at estimated radiation doses ranging from 100 to 700 rad (1–7 Gy) (Gomes et al. 1990). Some individuals exposed in the same incident exhibited typical radiation-induced skin lesions; the forearm was amputated in one individual with severe radiation injury (Brandão-Mello et al. 1991; Gomes et al. 1990).

No reports were located regarding dermal effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Ocular Effects.

Stable Cesium. No reports were located regarding ocular effects in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

Radioactive Cesium. No reports were located regarding ocular effects in humans following intermediate- or chronic-duration oral exposure to radioactive cesium. Among 20 patients hospitalized following acute external and internal (oral and dermal) exposure to a ^{137}Cs source, a few patients complained of lacrimation, hyperemia and edema of the conjunctiva, and ocular pain (Brandão-Mello et al. 1991). A few cases of protracted reduction in visual capacity were also reported, among which retinal injury was documented. In these cases, there was no change in lens transparency. These effects were due to the radiation, not to cesium *per se*.

No reports were located regarding ocular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Body Weight Effects.

Stable Cesium. No reports were located regarding body weight effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding body weight effects in animals following acute- or chronic-duration oral exposure to stable cesium. One intermediate-duration study found no significant effect on maternal body weight in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

Radioactive Cesium. No reports were located that associate body weight effects in humans or animals with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium compounds.

3.2.2.3 Immunological and Lymphoreticular Effects

Stable Cesium. No reports were located regarding immunological or lymphoreticular effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding immunological or lymphoreticular effects in animals following acute- or chronic-duration oral exposure to stable cesium. In one study of intermediate-duration, no significant effect on maternal spleen weight was noted in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

Radioactive Cesium. No reports were located that associate immunological or lymphoreticular effects in humans with intermediate- or chronic-duration oral exposure to radioactive cesium. Severe bone marrow depression, characterized by a low white blood cell count and consequent immunodeficiency, developed in 14 patients hospitalized following acute external and internal (oral and/or dermal) exposure to a ^{137}Cs source resulting in estimated absorbed doses ranging from 100 to 700 rad (1–7 Gy) (Brandão-Mello et al. 1991).

No reports were located regarding immunological or lymphoreticular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, severe bone marrow depression was observed in dogs exposed to ^{137}Cs by intravenous injection at activity levels resulting in estimated total bone marrow doses of 700–2,400 rad (7–24 Gy) (Nikula et al. 1995). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

The highest NOAEL values and all reliable LOAEL values for immunological and lymphoreticular effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-2 and plotted in Figure 3-2, since the same effects would be expected to occur in dogs following oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

3.2.2.4 Neurological Effects

Stable Cesium. No reports were located regarding neurological effects in humans following acute- or chronic-duration oral exposure to stable cesium. In one study of intermediate-duration exposure, an investigator, who voluntarily ingested about 34 mg Cs/kg/day (as cesium chloride) after morning and evening meals for 36 days, experienced general feelings of well-being, heightened sense perception, and tingling sensations in lips, cheeks, hands, and feet within 15 minutes of intake (Neulieb 1984). No self-reported adverse effects were noted in performance of mathematical tasks or in automobile driving skill.

No reports were located regarding neurological effects in animals following intermediate- or chronic-duration oral exposure to stable cesium. Rats administered unspecified acute gavage doses of cesium hydroxide exhibited initial signs of hyperexcitability followed by apathy and weakness during the course of 14 days of postdosing observation (Johnson et al. 1975).

Radioactive Cesium. No reports were located for humans or animals that associate neurological effects with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.2.2.5 Reproductive Effects

Stable Cesium. No reports were located regarding reproductive effects in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

Radioactive Cesium. No reports were located that associate reproductive effects in humans with intermediate- or chronic-duration oral exposure to radioactive cesium. Spermatozoa were reduced or absent in the semen of nine males examined approximately 1 month following presumed acute radiation doses on the order of several hundred rad from a ^{137}Cs source (Brandão-Mello et al. 1991). Exposures may have been both external and internal (via oral and dermal routes).

No reports were located regarding reproductive effects in animals following intermediate- or chronic-duration oral exposure to radioactive cesium. Significantly reduced fertility, expressed as the percentage of matings resulting in pregnancy (percent effective matings), was noted in male mice following a single oral administration of ^{137}Cs (as cesium nitrate) at activity levels that resulted in a total dose to the testis of approximately 300 rad (3 Gy) (Ramaiya et al. 1994). Complete, though temporary, sterility was evident at week 6 postadministration. By week 17 posttreatment, there were no significant differences in fertility between treatment and control groups. No significant reduction in male fertility was observed at activity levels resulting in total testicular radiation ranging from 10 to 100 rad (0.1–1 Gy). Significantly reduced fertility was also evident in male mice administered daily oral doses of ^{137}Cs (as cesium nitrate) for 2 weeks that resulted in total testicular radiation doses of about 385 rad (3.85 Gy), measured at 5 weeks posttreatment (Ramaiya et al. 1994).

Germinal epithelium damage and azoospermia were reported in dogs that had been administered ^{137}Cs (as cesium chloride) by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 742 to 1,640 rad (7.42–16.40 Gy) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

The highest NOAEL values and all reliable LOAEL values for reproductive effects from oral exposure to radioactive cesium are presented in Table 3-2 and plotted in Figure 3-2. The highest NOAEL values and all reliable LOAEL values for systemic effects in dogs exposed to ^{137}CsCl via intravenous injection are

also recorded in Table 3-2 and plotted in Figure 3-2, since the same effects would be expected to occur in dogs following oral exposure to ^{137}CsCl at levels that would result in comparable ^{137}Cs blood levels.

3.2.2.6 Developmental Effects

Stable Cesium. No reports were located regarding developmental effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding developmental effects in animals following acute- or chronic-duration oral exposure to stable cesium. In intermediate-duration studies, one investigator reported reduced body weight in offspring of mouse dams consuming approximately 115 mg Cs/kg/day during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b). Other observations included slight, but significant, changes in some organ weights and slight differences in activity of some hepatic enzymes among offspring of treated dams, relative to controls. Similar results were reported in the offspring of female mice consuming approximately 40 mg Cs/kg/day only during lactation (Messiha 1989b). However, gross and histopathologic examinations of the offspring, typical of well-designed developmental toxicity studies, were not performed in either of these studies, making them of little value for assessment of the developmental toxicity potential of cesium.

Radioactive Cesium. No reports were located that associate developmental effects in humans or animals with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.2.2.7 Cancer

No reports were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration exposure to stable or radioactive cesium. However, benign and malignant neoplasms were found in a variety of tissues and organs of dogs administered single intravenous doses of ^{137}Cs (as cesium chloride), which resulted in initial body burdens ranging from 1.0 to 4.0 mCi/kg (37–147 MBq/kg) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.2.5 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

All CEL values for dogs exposed to ^{137}CsCl via intravenous injection are recorded in Table 3-2 and plotted in Figure 3-2, since the same effect would be expected to occur in dogs following oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

3.2.3 Dermal Exposure

3.2.3.1 Death

Stable Cesium. No reports were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

Radioactive Cesium. There are no reports of deaths in humans that could be exclusively associated with acute-, intermediate-, or chronic-duration dermal exposure to radioactive cesium. As discussed in Section 3.2.2.1, several deaths occurred in humans following external and internal (oral and dermal) exposure to radioactive cesium.

There are no reports of deaths in animals following acute-, intermediate-, or chronic-duration dermal exposure to radioactive cesium.

3.2.3.2 Systemic Effects

Acute radiation syndrome, characterized by nausea, vomiting, and diarrhea was reported in a number of individuals exposed externally and internally (oral and/or dermal exposure) to a ^{137}Cs source in Goiânia, Brazil; other adverse effects included skin lesions, ocular lesions, severe bone marrow depression, and mild elevations in the activities of some liver enzymes (Brandão-Mello et al. 1991; Gomes et al. 1990; Rosenthal et al. 1991). See Section 3.2.2.2 for a more detailed description of systemic effects following mixed external and internal exposure to radioactive cesium.

No other reports were located regarding the following systemic effects in humans or animals following dermal exposure to stable or radioactive cesium: respiratory effects, cardiovascular effects, gastrointestinal effects, hematological effects, musculoskeletal effects, hepatic effects, renal effects, endocrine effects, body weight effects, metabolic effects.

Dermal Effects.

Stable Cesium. No reports were located regarding dermal effects in humans following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

No reports were located regarding dermal effects in animals following intermediate- or chronic-duration dermal exposure to stable cesium. Cesium hydroxide was considered a nonirritant on intact skin and a mild irritant on abraded skin of rabbits 24 and 48 hours, respectively, following closed-patch application of a 5% solution; similar application of cesium iodide resulted in no observed irritation (Johnson et al. 1975).

Radioactive Cesium. No reports were located regarding dermal effects in humans or animals that could be associated with dermal exposure to radioactive cesium. As discussed in Section 3.2.2.2, orofacial and dermal lesions were reported in a number of individuals following external and internal (oral and dermal) exposure to radioactive cesium (Brandão-Mello et al. 1991; Gomes et al. 1990).

Ocular Effects. No reports were located regarding eye irritation in humans resulting from ocular contact with stable or radioactive cesium.

A 5% solution of cesium hydroxide was extremely irritating and caustic to the rabbit eye (Johnson et al. 1975). It is likely that this effect was the result of the caustic nature of the hydroxide rather than an effect due to cesium *per se*. Similar treatment with cesium iodide resulted in no evidence of ocular irritation.

3.2.3.3 Immunological and Lymphoreticular Effects

Stable Cesium. No reports were located regarding immunological or lymphoreticular effects in humans following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

No reports were located regarding immunological or lymphoreticular effects in animals following acute- or chronic-duration dermal exposure to stable cesium. There was no indication of a sensitization response in guinea pigs following repeated intracutaneous injections of 0.1% solutions of cesium hydroxide or cesium iodide (Johnson et al. 1975).

Radioactive Cesium. No reports were located regarding immunological or lymphoreticular effects in humans or animals that could be associated with dermal exposure to radioactive cesium. As discussed in Section 3.2.2.3, severe bone marrow depression developed in 14 patients who were hospitalized following external and internal (oral and/or dermal) exposure to radioactive cesium (Brandão-Mello et al. 1991).

3.2.3.4 Neurological Effects

No reports were located for humans or animals that associate neurological effects with acute-, intermediate-, or chronic-duration dermal exposure to stable or radioactive cesium.

3.2.3.5 Reproductive Effects

Stable Cesium. No reports were located regarding reproductive effects in humans or animals following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

Radioactive Cesium. No reports were located that associate reproductive effects in humans with intermediate- or chronic-duration dermal exposure to radioactive cesium. Spermatozoa were reduced or absent in the semen of nine males examined approximately 1 month following acute overexposure to a ^{137}Cs source (Brandão-Mello et al. 1991). Exposures were both external and internal (via oral and dermal routes).

No reports were located regarding reproductive effects in animals following acute-, intermediate- or chronic-duration dermal exposure to radioactive cesium.

3.2.3.6 Developmental Effects

No reports were located that associate developmental effects in humans or animals with acute-, intermediate-, or chronic-duration dermal exposure to stable or radioactive cesium.

3.2.3.7 Cancer

No reports were located that associate cancer in humans or animals with acute-, intermediate-, or chronic-duration dermal exposure to stable or radioactive cesium.

3.2.4 External Exposure

This section contains information regarding health effects related to external exposure to radioactive cesium sources. Radionuclides of cesium, such as ^{137}Cs or ^{134}Cs, emit both beta particles and gamma rays, which are a health hazard in living organisms because they are capable of causing the ionization of atoms that they encounter. Beta particles can travel appreciable distances in air, but travel only a few millimeters in solids. External exposure to beta particles may result in damage to skin and superficial body tissues, but is not a threat to internal organs unless the radiation source is internalized. Gamma radiation, on the other hand, can easily pass completely through the human body and cause ionization of atoms in its path. Several feet of concrete or a few inches of lead shielding are required for protection from gamma rays. Because it is so highly penetrating, external gamma radiation released by radionuclides such as cesium is a radiation hazard to internal organs (ATSDR 1999; EPA 1998).

The purpose of this section is to provide information regarding health effects associated with external exposure to a radioactive cesium source. External exposure to radioactive cesium is simply exposure to beta and gamma radiation; there is nothing unique to cesium *per se*. The same hazards exist from external exposure to any source of beta and gamma radiation. Refer to ATSDR (1999) for a detailed description of health effects from external exposure to ionizing radiation in general.

3.2.4.1 Death

There are no reports of deaths in humans that could be exclusively associated with acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Death was noted within a few weeks following acute external and internal (oral and dermal) exposure to a ^{137}Cs source that resulted in estimated radiation doses ranging from 400 to 600 rad (4–6 Gy) (Brandão-Mello et al. 1991). See Section 3.2.2.1 for more detailed information.

Significantly reduced survival was noted in rat fetuses following whole-body irradiation (via a ^{137}Cs source) of pregnant dams on gestational day 14 at acute radiation doses $400 rad (4.0 Gy); an LD_{50} value

was about 500 rad (5.0 Gy) (Koshimoto et al. 1994). No reports were located regarding death in animals following intermediate- or chronic-duration external exposure to radioactive cesium.

3.2.4.2 Systemic Effects

Acute radiation syndrome, characterized by nausea, vomiting, and diarrhea, was reported in a number of individuals exposed externally and internally (oral and/or dermal exposure) to a ^{137}Cs source in Goiânia, Brazil; other adverse effects included skin lesions, ocular lesions, severe bone marrow depression, and mild elevations in the activities of some liver enzymes (Brandão-Mello et al. 1991; Gomes et al. 1990; Rosenthal et al. 1991). See Section 3.2.2.2 for a more detailed description of systemic effects following mixed exposure to radioactive cesium.

No other reports were located regarding the following systemic effects in humans or animals that could be exclusively associated with external exposure to stable or radioactive cesium: respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, endocrine, dermal, ocular, body weight, metabolic, and other systemic effects.

3.2.4.3 Immunological and Lymphoreticular Effects

No reports were located that associate immunological or lymphoreticular effects in humans with intermediate- or chronic-duration external exposure to radioactive cesium. Severe bone marrow depression, characterized by neutropenia and thrombocytopenia, developed in 14 patients hospitalized following acute external and internal (oral and dermal) exposure to a ^{137}Cs source resulting in estimated absorbed doses ranging from 100 to 700 rad (1.0–7 Gy) (Brandão-Mello et al. 1991). This effect was the result of the radiation, not the presence of cesium *per se*.

No reports were located regarding immunological or lymphoreticular effects in animals following acute-, intermediate-, or chronic-duration external exposure to radioactive cesium.

3.2.4.4 Neurological Effects

No reports were located that associate neurological effects in humans or animals with postnatal acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Neurological effects associated with *in utero* exposure are discussed in Section 3.2.4.6 (Developmental Effects).

3.2.4.5 Reproductive Effects

No reports were located that associate reproductive effects in humans with intermediate- or chronic-duration external exposure to radioactive cesium. Spermatozoa were reduced or absent in the semen of nine males examined approximately 1 month following presumed acute exposure to a ^{137}Cs source (Brandão-Mello et al. 1991). Exposures were both external and internal (via oral and dermal routes).

No reports were located regarding reproductive effects in animals following acute- or chronic-duration external exposure to radioactive cesium. Significantly reduced fertility, expressed as the percentage of matings resulting in pregnancy (% effective matings), was noted in male mice following external exposure to a ^{137}Cs source 23 hours a day for 19.5 days at a dose rate of 6.75 mrad/hour (0.675 mGy/hour), resulting in a total dose of 300 rad (3 Gy) (Ramaiya et al. 1994). Complete sterility was evident in males by the third week following exposure termination. During weeks 1 and 2 posttreatment, significantly increased total and postimplantation embryo mortality was noted. These effects were the result of the radiation, not the presence of cesium *per se*.

The highest NOAEL values and all reliable LOAEL values for reproductive effects from external exposure to radioactive cesium are presented in Table 3-3 and plotted in Figure 3-3.

3.2.4.6 Developmental Effects

No reports were located that associate developmental effects in humans with acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Cells of the developing central nervous system are among the most sensitive to the effects of ionizing radiation in the developing fetus (ATSDR 1999). Schull and Otake (1999) cite numerous reports in which impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed (during weeks 8–15 or 16–25 postovulation) to ionizing radiation from the bombs. The small number (38) of mentally-impaired survivors makes it difficult to generalize a dose-response relationship. However, the data are compatible

Table 3-3. Levels of Significant Exposure to Cesium - Radiation Toxicity - External radiation

	Species (Strain)	Exposure/ Duration/ Frequency	System	NOAEL (rad)	LOAEL Less serious (rad)		LOAEL Serious (rad)		Reference Chemical Form
	ACUTE EXPOSURE								
	Developmental								
1	Rat (Wistar)	Once Gd 13, 14, or 15			50	(increased micronuclei in hematocytes)	400	(31.5% fetal mortality)	Koshimoto et al. 1994 ^{137}Cesium
2	Rat (Sprague-Dawley)	Once Gd 11 or 17					100	(altered motor function, decreased cortical thickness)	Norton and Kimler 1987 ^{137}Cesium
3	Rat (Sprague-Dawley)	Once Gd 13, 15, or 17					75	(altered motor function, decreased cortical thickness, most prominent when exposed on Gd 15)	Norton and Kimler 1988 ^{137}Cesium
4	Mouse (C57BL/6)	Once Gd 14			100 M	(6% lower body weight, 9% lower brain weight, reduced brain size)			Minamisawa et al. 1990 ^{137}Cesium
5	Mouse (C57BL/6)	Once Gd 14			100 M	(16% lower body weight, increased aggressive behavior)			Minamisawa et al. 1992 ^{137}Cesium
6	Mouse Swiss albino	Once Gd 12			400	(retarded odontogenesis)			Saad et al. 1991 ^{137}Cesium
7	Mouse Swiss albino	Once Gd 12			400	(smaller litter size, smaller head size, delayed palatogenesis)			Saad et al. 1994 ^{137}Cesium

Table 3-3. Levels of Significant Exposure to Cesium - Radiation Toxicity - External radiation (continued)

	Species (Strain)	Exposure/ duration/ frequency	System	NOAEL (rad)	LOAEL Less serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	Cancer						
8	Rat WAG/Rij	Once at 8, 12, 16, 22, 36, or 64 wk of age				100 F (mammary carcinoma in 57/200 rats)	Bartstra et al. 1998 ^{137}Cesium
	INTERMEDIATE EXPOSURE						
	Reproductive						
9	Mouse (Hybrid)	19.5 d 23 hr/d				300 M (reduced fertility)	Ramaiya et al. 1994 ^{137}Cesium

*The number corresponds to entries in Figure 3-3.

d = day(s); F = female; Gd = gestational day; hr = hour(s); LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; wk = week(s).

Figure 3-3. Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation

Acute (≤14 days)

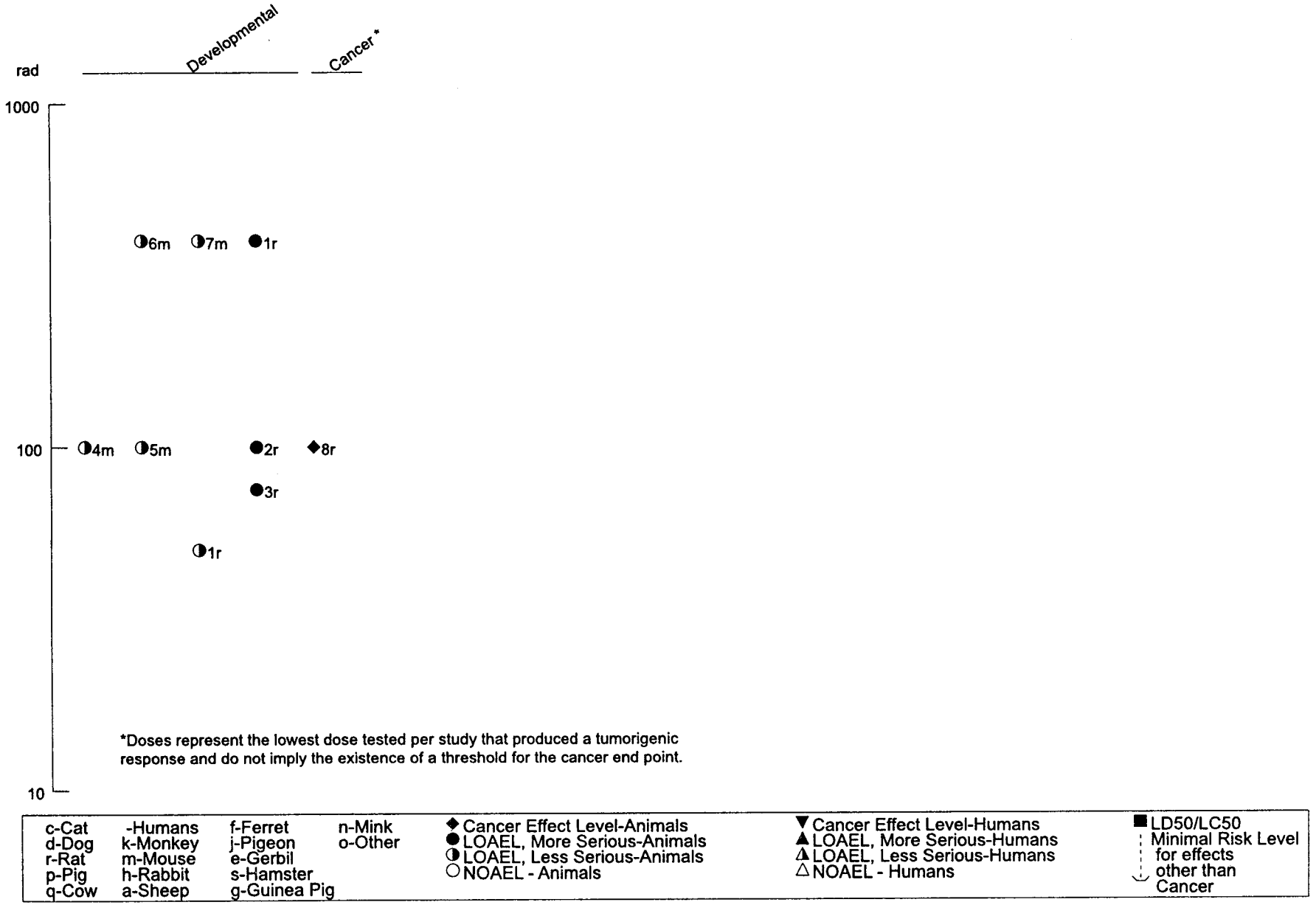


Figure 3-3. Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation (*continued*)

Intermediate (15-364 days)

with either a threshold dose of 20–40 rad (0.2–0.4 Gy) or zero threshold linear response. Although such effects would be expected in individuals exposed to similar levels of external radiation from any source of gamma radiation, it is unlikely that such high levels would be achieved from any radiocesium source.

No reports were located regarding developmental effects in animals following intermediate- or chronic-duration external exposure to radioactive cesium. Significantly reduced postnatal body weight, impaired motor activity, and decreased thickness within cortical layers of the brain were observed in young rats (7–21 days postpartum) exposed during gestation by whole-body radiation of pregnant dams via a ^{137}Cs source at a rate of approximately 50 rad/minute (0.5 Gy/minute) for a total radiation dose of 75 or 100 rad (0.75 or 1 Gy) (Norton and Kimler 1987, 1988). The effects were of larger magnitude when the fetal rats were exposed on gestation day 15 rather than earlier (gestation days 11 or 13) or later (gestation day 17). Significantly smaller litter size, smaller head size, and retarded odontogenesis were observed in fetuses of pregnant mice exposed on gestation day 12 to whole-body irradiation from a ^{137}Cs source for 4.9 minutes (resulting in a total radiation dose of 400 rad or 4 Gy) and examined on gestation day 18. Additionally, all exposed fetuses exhibited cleft palate on examination while normal closure was observed in all unexposed control fetuses (Saad et al. 1991, 1994). Significantly reduced survival was observed in rat fetuses following whole-body irradiation (via a ^{137}Cs source) of pregnant dams on gestation day 14 at acute radiation doses of 400 rad (4.0 Gy) or greater. An LD_{50} value for fetuses was about 500 rad (5.0 Gy) (Koshimoto et al. 1994). Aggressive behavior was studied in male mice (100–135 days of age) exposed *in utero* on gestation day 14 through whole-body irradiation of pregnant dams via an external ^{137}Cs source at total radiation doses of 100 or 200 rad (1 or 2 Gy) (Minamisawa et al. 1992). Incidences of aggressive behavior were significantly higher among irradiated groups, relative to untreated controls. The intensity of aggressive behavior was significantly higher only in the 200 rad (2 Gy) exposure group. Minamisawa et al. (1990) found dose-related significantly decreased brain weight in 6-month-old mice that had been irradiated on gestation day 14 at doses of 100–300 rad (1–3 Gy). In each of these studies (Koshimoto et al. 1994; Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988), the observed developmental effects were the result of radiation exposure, not the presence of cesium *per se*.

Kusama and Hasegawa (1993) designed a study to examine the relationship between fetal developmental stage at the time of external exposure to gamma rays from a ^{137}Cs source and the occurrence of external malformations and growth retardation. Groups of pregnant ICR mice were irradiated once with 150 rad (1.5 Gy) at a dose rate of 20 rad/minute (0.2 Gy/minute) on 6-hour intervals during the period of organogenesis (gestation days 6.5–14). Fetuses were examined on gestation day 18. The authors reported peaks in the occurrence of exencephaly among fetuses irradiated during gestation days

6.5–8.75 and 10.25–10.75, and the highest peak occurred at gestation day 7.5. Peaks in the occurrence of cleft palate were seen in fetuses irradiated at gestation days 8.75 and 10.75. The most apparent reduction in body weight of irradiated fetuses, relative to controls, occurred in groups irradiated between gestation days 9.75 and 11. The observed developmental effects were the result of radiation exposure, not the presence of cesium *per se*.

The highest NOAEL values and all reliable LOAEL values for developmental effects from external exposure to radioactive cesium are presented in Table 3-3 and plotted in Figure 3-3.

3.2.4.7 Cancer

No reports were located regarding cancer in humans following acute-, intermediate-, or chronic-duration external exposure to radioactive cesium in particular. Due to the nature of ionizing radiation in general, carcinogenic effects similar to those observed in Japanese survivors of the 1945 atomic bombing incidents might be expected among individuals acutely exposed to high levels of radiation from a radioactive cesium source. In the only available report of high-level exposure specifically to radioactive cesium (^{137}Cs, the accidental human exposures in Goiânia, Brazil in 1987), the incident is too recent for meaningful data on the potential for carcinogenicity. No reports were located in which increased cancer risk could be associated with long-term exposure to low levels of ionizing radiation.

No reports were located regarding cancer in animals following intermediate- or chronic-duration external exposure to radioactive cesium. Increased lifetime risk of mammary tumors was observed in female rats exposed to single whole-body doses of 100 or 200 rad (1 or 2 Gy) of ^{137}Cs radiation at a dose rate of 75 rad/minute (0.75 Gy/minute) between the ages of 8 and 36 weeks (Bartstra et al. 1998). The excess normalized risk values for carcinoma were 0.9 and 2.2 for 100 and 200 rad (1 and 2 Gy) doses, respectively, with no significant differences between the age groups of 8, 12, 16, 22, or 36 weeks. Irradiation at 64 weeks, however, yielded fewer carcinomas than unirradiated controls. The excess normalized risk values were found to be -0.7 and -0.3 for 100 and 200 rad (1 and 2 Gy) doses, respectively. These effects were the result of the radiation, not the presence of cesium *per se*.

3.2.5 Other Routes of Exposure

Although parenteral injection is not considered to be an exposure route of concern for the general population, it has been considered to be a good indicator of adverse health effects that would be expected in laboratory animals following the absorption of ^{137}CsCl into the blood if such animals were to be exposed via inhalation or oral routes (Boecker et al. 1969a; Melo et al. 1996, 1997; Nikula et al. 1995, 1996). This argument is based on the results of biokinetic studies in dogs (Boecker et al. 1969a) in which intravenous injections of ^{137}CsCl resulted in temporal and tissue distribution patterns and tissue doses of ^{137}Cs that were similar to those resulting from inhalation exposure (Boecker et al. 1969a). Similar tissue distribution and retention kinetics were also shown in guinea pigs whether exposure to ^{137}CsCl had been via intraperitoneal, inhalation, or oral routes (Stara 1965).

In dogs, intravenous administration of soluble ^{137}CsCl has resulted in depression of a number of blood factors, severe bone marrow depression, germinal cell damage (males), and early death (Nikula et al. 1995, 1996; Redman et al. 1972). Long-term surviving dogs exhibited increased incidences of benign and malignant neoplasms in a variety of tissues and organs, with no apparent single target organ of toxicity.

3.2.5.1 Death

Dose-related decreased survival was observed in young adult beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens of 1.9–3.8 mCi/kg (71.7–141 MBq/kg) (Nikula et al. 1995). All six dogs in the highest exposure group died between 19 and 33 days post injection. The total whole body radiation dose to death in this group of dogs averaged 1,180 rad (11.8 Gy). Deaths were attributed to severe pancytopenia resulting from hematopoietic cell damage. In a study of 63 other beagle dogs, intravenous injection of ^{137}CsCl resulted in initial body burdens of approximately 1.7–4.0 mCi/kg (61–147 Mbq/kg) (Nikula et al. 1996). These dogs, grouped according to age, were juveniles (142–151 days old), young adults (388–427 days old), or middle-aged adults (1,387–2,060 days old) at the time of injection. Early mortality, within 52 days post injection, was noted in 10/10 middle-aged dogs, 10/38 young adults, and 3/15 juveniles. Average initial ^{137}Cs body burdens among the early deaths were in the range of 3.6–4.0 mCi/kg (133–147 MBq/kg). The middle-aged dogs died significantly earlier ($p=0.002$) than the juvenile or young adult dogs, and middle-aged female dogs died significantly earlier ($p=0.002$) than middle-aged male dogs.

All reliable LOAEL values for death in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Tables 3-1 and 3-2 and plotted in Figures 3-1 and 3-2, since the same effect would be expected to occur in dogs following inhalation or oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

3.2.5.2 Systemic Effects

No data were located regarding respiratory effects, gastrointestinal effects, musculoskeletal effects, hepatic effects, renal effects, endocrine effects, dermal effects, ocular effects, body weight effects, or metabolic effects in humans or animals following exposure to stable or radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

The highest NOAEL values and all reliable LOAEL values for systemic effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Tables 3-1 and 3-2 and plotted in Figures 3-1 and 3-2, since the same effects would be expected to occur in dogs following inhalation or oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

Cardiovascular Effects. Cardiac arrhythmias were elicited in experimental animals following the injection of stable cesium chloride directly into the circulatory system (Brachmann et al. 1983; Levine et al. 1985; Murakawa et al. 1997; Patterson et al. 1990).

Hematological Effects. Hematological dyscrasia, characterized by severe thrombocytopenia and leukopenia, and death within 81 days were observed in 11 of 54 dogs that had been administered single intravenous injections of ^{137}CsCl (Nikula et al. 1995; Redman et al. 1972). The early deaths occurred in groups with average initial body burdens ranging from 1.9 to 3.8 mCi (71.7–141 MBq/kg) and cumulative doses to death in the range of 1,180–1,400 rad (11.8–14.0 Gy). Moderately to severely depressed blood cell counts were observed among 25 surviving dogs, some of which had been injected with lower levels of ^{137}CsCl that resulted in average initial body burdens of 1.0 or 1.4 mCi/kg (36.4 or 51.7 MBq/kg). Other treatment-related hematological effects included bone marrow aplasia, decreased platelet levels, and reduced packed-cell volume. In long-term surviving dogs, depressed blood values returned toward normal within during the first year post treatment. Severely depressed blood cell counts were observed in 23 dogs that died within 52 days following single intravenous administration of ^{137}CsCl at levels resulting in initial body burdens in the range of 1.7–4.4 mCi/kg (61–162 MBq/kg) (Nikula et al. 1996).

3.2.5.3 Immunological and Lymphoreticular Effects

Severe bone marrow depression was observed in dogs administered ^{137}CsCl by intravenous injection at activity levels resulting in estimated total bone marrow doses of 700–2,400 rad (7–24 Gy) (Nikula et al. 1995).

The highest NOAEL values and all reliable LOAEL values for immunological and lymphoreticular effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Tables 3-1 and 3-2 and plotted in Figures 3-1 and 3-2, since the same effects would be expected to occur in dogs following inhalation or oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

3.2.5.4 Neurological Effects

No data were located regarding neurological effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

3.2.5.5 Reproductive Effects

Persistent germinal epithelium damage and azoospermia were reported in all long-term surviving dogs that had been administered ^{137}CsCl by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 742 to 1,640 rad (7.42–16.40 Gy) (Nikula et al. 1995, 1996).

The highest NOAEL values and all reliable LOAEL values for systemic effects in dogs exposed to ^{137}CsCl via intravenous injection are recorded in Tables 3-1 and 3-2 and plotted in Figures 3-1 and 3-2, since the same effects would be expected to occur in dogs following inhalation or oral exposure to ^{137}CsCl at levels that would result in comparable ^{137}Cs blood levels.

3.2.5.6 Developmental Effects

No data were located regarding developmental effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

3.2.5.7 Cancer

Benign and malignant neoplasms were found in a variety of tissues and organs of beagle dogs that had been administered single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996). Fifty-four young adult dogs at the Inhalation Toxicology Research Institute (ITRI) received amounts of ^{137}CsCl that resulted in average initial body burdens ranging from 1.0 to 3.8 mCi/kg (37–141 MBq/kg) (Nikula et al. 1995). In a study initiated at the Argonne National Laboratory (ANL), 63 beagle dogs, grouped according to age at study initiation (juveniles, 142–151 days old; young adults, 388–427 days old; middle-aged adults, 1,387–2,060 days old), were administered single intravenous injections of ^{137}CsCl that resulted in initial body burdens of approximately 1.7–4.0 mCi/kg (61–147 MBq/kg) (Nikula et al. 1996). In both studies, dose-related increased incidences were observed for malignant neoplasms, malignant neoplasms excluding mammary neoplasms, all sarcomas considered as a group, all nonmammary carcinomas considered as a group, and malignant liver neoplasms. An increased risk for malignant thyroid neoplasms was seen in the ANL male dogs, but not in the ITRI males or females. In the ITRI (but not ANL) dogs, an increased relative risk for benign neoplasms excluding mammary neoplasms was noted. The occurrence of neoplasms in a diversity of tissues and organs might reflect the widespread distribution of cesium in the body.

All CEL values for dogs exposed to ^{137}CsCl via intravenous injection are recorded in Tables 3-1 and 3-2 and plotted in Figures 3-1 and 3-2, since the same effect would be expected to occur in dogs following inhalation or oral exposure to ^{137}CsCl levels that would result in comparable ^{137}Cs blood levels.

3.3 GENOTOXICITY

Genotoxicity data for stable and radioactive cesium are summarized in Tables 3-4 and 3-5, respectively.

Evidence for genotoxic effects of stable cesium is limited to studies in which cesium chloride induced dose-dependent significantly increased incidences of chromosomal aberrations in human lymphocytes *in vitro* (Ghosh et al. 1993) (see Table 3-4) and mouse bone marrow *in vivo* (Ghosh et al. 1990, 1991) (see Table 3-5). Cesium sulfate was not mutagenic in *Escherichia coli (E. coli)* tester strains PQ37 and PQ35 either with or without metabolic activation in the SOS Chromotest (a bacterial colorimetric assay) at doses up to those resulting in significant toxicity (Olivier and Marzin 1987).

Table 3-4. Genotoxicity of Cesium *In Vitro*

Species (test system)	End point	Result: With activation	Result: Without activation	Reference
Stable Cesium				
Mammalian cells:				
Human lymphocytes	Chromosomal aberrations	ND	+	Ghosh et al. 1990, 1991
Prokaryotic organisms:				
Escherichia coli (PQ 37 and PQ 35)	Mutations	–	–	Olivier and Marzin 1987
Radioactive Cesium				
Mammalian cells:				
Human peripheral blood lymphocytes	Micronuclei	ND	+	Balasem and Ali 1991
Human peripheral blood lymphocytes	Chromosomal aberrations	ND	+	Doggett and McKenzie 1983
Human peripheral blood lymphocytes	Chromosomal aberrations	ND	+	Hintenlang 1993
Human peripheral blood lymphocytes	Chromosomal aberrations	ND	+	Iijima and Morimoto 1991
Human peripheral blood lymphocytes	Sister chromatid exchange	ND	–	Iijima and Morimoto 1991
Human spermatozoa	Chromosomal aberrations	ND	+	Mikamo et al. 1990, 1991

Table 3-4. Genotoxicity of Cesium *In Vitro (continued)*

Species (test system)	End point	Result		Reference
		With activation	Without activation	
Radioactive Cesium (*cont.*)				
Mammalian cells (*cont.*):				
Human spermatozoa and zona-free hamster oocytes fertilization system	Micronuclei	ND	+	Kamiguchi et al. 1991
Mouse (BALB/c, SC3T3/W, Scid/St cells)	DNA double-strand breaks	ND	+	Biedermann et al. 1991
Chinese hamster ovary cells	Chromosomal aberrations, sister chromatid exchange	ND	+	Arslan et al. 1986

DNA = deoxyribonucleic acid; ND = no data; – = negative results; + = positive results

Table 3-5. Genotoxicity of Cesium *In Vivo*

Species (test system)	End point	Results	Reference
Stable Cesium			
Mammalian cells:			
Mouse bone marrow	Chromosomal aberrations	+	Ghosh et al. 1993
Radioactive Cesium			
Mammalian cells:			
Human peripheral blood lymphocytes	Chromosomal aberrations	+	Natarajan et al. 1998
Human peripheral blood lymphocytes	Chromosomal aberrations	+	Padovani et al. 1993
Monkey germ cells (male)	Reciprocal translocations	+	Tobari et al. 1988
Monkey germ cells (male)	Reciprocal translocations	+	Ramaiya et al. 1994
Mouse germ cells (male)	Reciprocal translocations	+	Ramaiya et al. 1994
Mouse germ cells (male)	Dominant lethal mutations	+	Ramaiya et al. 1994

+ = positive results

Increased frequency of point mutations in T-lymphocytes was observed in individuals in Goiânia, Brazil who had been exposed to a ^{137}Cs source approximately 2.5 years prior to testing (Skandalis et al. 1997). The estimated dose from external radiation was 170 rad (1.7 Gy). The authors estimated internal doses, based on whole-body counts and measured activity in urine and feces. However, realistic estimates were not reported. Among individuals exposed in the same incident, frequencies of chromosomal aberrations were used to estimate external radiation doses (Natarajan et al. 1998). No human reports were located in which genotoxic effects could be associated with specific radiation exposure levels, nor was there any information regarding potential for route-specific differences in observed genotoxic effects related to radioactive cesium exposure. Five years after the initial exposure to radioactive fallout from the Chernobyl accident of 1986, slightly greater frequencies of chromosomal aberrations were observed in peripheral blood lymphocytes of three groups of Byelorussian children (41 total) living in areas contaminated with ^{137}Cs fallout than in those of an Italian control group of 10 children (Padovani et al. 1993). Whole-body counts found internally deposited ^{137}Cs activity ranges of 12–75 nCi (0.46–2.8 kBq) in children from Navrovl'a, an area (70 km from Chernobyl) exhibiting ^{137}Cs contamination of 14–40 Ci/km^2 (518 GBq/km^2). Internally deposited ^{137}Cs activity ranges of 1.2–10.8 nCi (0.044–0.4 kBq) and 208–872 nCi (7.7–32.3 kBq) were reported for children evacuated from the Chernobyl area soon after the accident to areas 200–300 km from Chernobyl with ^{137}Cs ground contamination of 1–10 Ci/km^2 (37–370 GBq/km^2), and children living in the Stolin area (250 km from Chernobyl) with ^{137}Cs ground contamination of 1–5 Ci/km^2 (37–185 GBq/km^2), respectively. Internalized activity was presumably from the consumption of ^{137}Cs-contaminated food. Although a small increase in the frequency of chromosomal aberrations in lymphocytes was observed, no pathology was apparent. These genotoxic effects were the result of the radiation, not the presence of cesium *per se*.

A dose-related increased frequency of micronuclei was observed in human peripheral blood lymphocytes exposed *in vitro* to gamma radiation from a ^{137}Cs source at doses ranging from 5 to 600 rad (0.05–6.00 Gy) (Balasem and Ali 1991). This effect was the result of the radiation, not the presence of cesium *per se*.

In mice, genotoxic effects resulting from repeated oral exposure (daily administration for 2 weeks) to ^{137}Cs (as cesium nitrate) were compared with those elicited from external whole-body irradiation (23 hours/day for 19.5 days) using a ^{137}Cs source (Ramaiya et al. 1994). At comparable total radiation doses (approximately 300–400 rad or 3–4 Gy), both exposure scenarios resulted in similar increases in dominant lethal mutations among exposed male mice. Significant increases in the frequency of reciprocal translocations have been reported in spermatogonia of mice orally administered single doses of ^{137}Cs (as

cesium chloride) that resulted in absorbed total body doses of approximately 300 rad (3 Gy) (Ramaiya et al. 1994). These effects were the result of the radiation, not the presence of cesium *per se*.

Significant (dose-related) increases in the formation rate of micronuclei were seen in blood cells of fetal rats following irradiation of pregnant dams to total radiation doses of 50–400 rad (0.5–4 Gy), from a ^{137}Cs source, on gestation day 14 (Koshimoto et al. 1994). In crab-eating monkeys exposed to gamma radiation from an external ^{137}Cs source, increases in reciprocal translocations in spermatogonia were dose-related through the total absorbed dose range of 30–150 rad (0.3–1.5 Gy); it was also noted that the induction rate of translocations after acute high-dose-rate (25 rad/minute or 0.25 Gy/minute) exposure was about 10 times higher than that resulting from longer-term low-dose-rate ($1.8x10^{-5}$ rad/minute or $1.8x10^{-7}$ Gy/minute) exposure (Tobari et al. 1988). These effects were the result of the radiation, not the presence of cesium *per se*.

Additional assays, performed *in vitro*, have also indicated that radioisotopes of cesium are genotoxic; specific end points include chromosomal aberrations and breaks, sister chromatid exchanges, and micronuclei (Arslan et al. 1986; Biedermann et al. 1991; Doggett and McKenzie 1983; Hintenlang 1993; Iijima and Morimoto 1991; Kamiguchi et al. 1991; Mikamo et al. 1990, 1991). Refer to ATSDR (1999) for more information on the genotoxic effects of ionizing radiation.

3.4 TOXICOKINETICS

Numerous biokinetic studies have been performed in animals exposed internally to small amounts of the radioisotope ^{137}Cs. The biokinetic behavior of cesium has also been studied in humans either given tracer amounts of radiocesium or accidently exposed to larger amounts.

Inhaled or ingested cesium (in soluble compounds) is rapidly absorbed into the blood, and is distributed to all major tissues and organs. Insoluble particles that are ingested are mostly excreted in the feces. Human and animal studies indicate that only a small fraction of the ingested cesium is absorbed. Cesium that comes into contact with the skin may be absorbed to some extent through the skin.

Following uptake by the blood, widespread distribution of cesium to all major soft tissues is observed in humans and animals. Cesium levels are slightly higher in skeletal muscle than other tissues. Distribution patterns in animals have been shown to be similar following exposure by inhalation, oral, and parenteral

routes of exposure. Cesium crosses the placenta to the fetus. Cesium is also found in breast milk of a mother with an internal deposition.

Cesium behaves in a manner similar to potassium. Both cesium and potassium concentrate in intracellular fluid. Cesium can replace potassium in biological systems, and cesium retention has been experimentally estimated based on the retention of potassium.

Excretion rates for ^{137}Cs have been studied in numerous populations exposed to nuclear fallout following incidents such as the Chernobyl accident. Biologically based pharmacokinetic models have been developed to describe relationships between intake and elimination. Experimental human studies have also been performed using tracer amounts of ^{134}Cs and ^{137}Cs. Urinary excretion is the primary route of elimination of cesium, and is independent of the route of exposure. Urinary to fecal ratios for cesium in humans have been found to range from 2.5:1 to 10:1. Radiocesium excretion rates were lower in males with muscular dystrophy than in normal, age-matched controls, and higher in pregnant women than in others who were not pregnant (Bengtsson et al. 1964; Rundo and Turner 1966; Zundel et al. 1969).

The elimination of cesium in humans appears to be age- and sex-related and may be principally a function of body mass. Children ages 5–14 exhibited average elimination half-times for cesium of 20 days, with no significant difference between males and females; elimination half-times in older groups were significantly higher (47 days for adolescent and adult females; 67 days in 15-year-old males; 93 days in males 30–50 years of age) (Boni 1969b). Long-term retention also appears to be age related in dogs injected intravenously with $^{137}CsCl$; puppies 3–5 months of age exhibited elimination half-times that are shorter than those of adult dogs (Melo et al. 1997).

3.4.1 Absorption

3.4.1.1 Inhalation Exposure

Inhalation exposure to relatively soluble cesium compounds will result in absorption of cesium in humans, although no reports were located that measured absorption of cesium following inhalation exposure. Evidence for absorption was presented by Miller (1964) who reported information on whole-body counts of ^{137}Cs (taken periodically for up to 285 days) in two men following occupational exposure to ^{137}Cs (as cesium sulfate), that was presumed to have been by inhalation. Distribution of ^{137}Cs was

relatively uniform throughout the body, and steadily decreasing whole-body counts indicated that ^{137}Cs was eliminated from the body with a biological half-time of approximately 73–84 days.

Inhaled soluble cesium compounds are readily absorbed and distributed systemically in animals. Approximately 80% cesium absorption was observed in dogs acutely exposed to small amounts of aerosolized ^{137}Cs (as cesium chloride) (Boecker 1969a, 1969b). Deposition and distribution of cesium following inhalation exposure to radiolabeled cesium chloride was also observed in rats (Lie 1964; Stara and Thomas 1963) and guinea pigs (Stara 1965). Absorption was rapid following inhalation exposure.

3.4.1.2 Oral Exposure

It is generally accepted that cesium ingested in soluble cesium compounds is well absorbed by the gastrointestinal tract of humans and animals. Henrichs et al. (1989) reported an average cesium absorption percentage of 78% in a group of 10 adult (5 male, 5 female) volunteers ingesting a meal of venison that was highly contaminated with ^{137}Cs and ^{134}Cs. Further observations indicating that soluble cesium compounds were absorbed after ingestion by humans include: (1) low fecal excretory rates, (2) urinary excretory rates 4–10 times higher than those of fecal excretion, and (3) elimination half-times ranging from 45 to 147 days (Henrichs et al. 1989; Iinuma et al. 1965; Richmond et al. 1962; Rosoff et al. 1963).

Absorption of ^{137}Cs from ingestion of radioactive fallout particles was found to be in the range of only 3%, indicating that such particles are relatively insoluble in biological fluids (LeRoy et al. 1966). Measurable amounts of ^{137}Cs were found in breast milk of women living in areas contaminated with radioactive fallout from the Chernobyl nuclear accident (Johansson et al. 1998). Transfer of ^{137}Cs from the mother to a nursing newborn was estimated to be approximately 40%; for a 1-year-old child, the transfer percentage was somewhat higher (50%).

Animal studies support the findings in humans. Rapid absorption and widespread distribution of cesium was reported in guinea pigs administered single oral doses of soluble ^{137}Cs (as cesium chloride); fractional absorption data were not reported (Stara 1965). In rats orally administered single doses of highly insoluble irradiated fuel particles (mean diameter of 0.93 μm) containing ^{137}Cs and other radioactive elements, absorption of ^{137}Cs was found to be <10% (Talbot et al. 1993).

3.4.1.3 Dermal Exposure

Dermal absorption has been qualitatively demonstrated in rats (Pendic and Milivojevic 1966). Traces of ^{137}Cs were observed in the blood of rats within a few minutes following the dermal application of $^{137}CsCl$ in aqueous solution. Approximately 3% of $^{137}CsCl$ applied to a surface area of several cm^2 was absorbed within 6 hours.

3.4.2 Distribution

3.4.2.1 Inhalation Exposure

Once absorbed, cesium is rapidly distributed throughout the body. Separate measurements of radioactivity from head, chest, upper abdomen, lower abdomen, thighs, legs, and feet indicated that ^{137}Cs was widely distributed throughout the bodies of two men who were occupationally exposed to ^{137}Cs (Miller 1964). Proportions of radioactivity in these body segments remained relatively constant from days 9–285 postexposure, indicating that ^{137}Cs was not likely to have been selectively accumulating in a particular region.

Animal studies also indicate a relatively uniform distribution of cesium following inhalation exposure to soluble cesium compounds (Boecker 1969a, 1969b; Stara 1965). Within 2 hours postexposure to aerosols of ^{137}Cs (as cesium chloride), up to 60% of the total body burden of ^{137}Cs was found in tissues other than respiratory or gastrointestinal tracts of dogs (Boecker 1969b). Concentrations of ^{137}Cs in skeletal muscles, diaphragm, kidneys, and mandibular salivary gland were slightly higher than the whole-body average; concentrations in lung, skin, femur, fat, and blood were somewhat lower (Boecker 1969b). Other tissues exhibited concentrations approximating the whole-body average. A relatively uniform distribution of ^{137}Cs in numerous body tissues, with the highest concentrations in skeletal muscle, were also observed in guinea pigs and rats exposed by inhalation (Lie 1964; Stara 1965; Stara and Thomas 1963).

3.4.2.2 Oral Exposure

Widespread distribution of cesium was observed in humans following oral exposure to soluble cesium compounds. In two human subjects orally administered ^{137}Cs (as cesium chloride), whole blood levels of ^{137}Cs within the first hour postadministration amounted to approximately 2–3% of the amount

administered, indicating that ^{137}Cs was rapidly absorbed and well distributed via the circulation (Rosoff et al. 1963).

Animal studies also showed relatively uniform distribution following oral exposure to soluble cesium compounds. Guinea pigs exhibited ^{137}Cs in numerous body tissues after receiving single oral doses of ^{137}Cs (as cesium chloride). The highest concentrations were found in skeletal muscle (Stara 1965). After the first day postadministration, no significant differences in ^{137}Cs distribution patterns were observed between groups of guinea pigs exposed by inhalation, oral administration, or intraperitoneal injection of ^{137}Cs (as cesium chloride) (Stara 1965). Dogs and mice exhibited relatively uniform distribution of cesium throughout body tissues following chronic oral administration of ^{137}Cs (as cesium chloride) (Furchner et al. 1964). Cesium also crossed the placenta of animals and was found in breast milk. Newborn lambs exhibited lower tissue levels of ^{134}Cs than their mothers following oral administration of radiolabeled cesium chloride during pregnancy (Vandecasteele et al. 1989). The concentrations of ^{134}Cs in nursing lambs eventually exceeded the levels in their mothers.

3.4.2.3 Dermal Exposure

One report was located regarding distribution of cesium in animals following dermal exposure (Pendic and Milivojevic 1966). The investigators found rapid absorption and widespread distribution of ^{137}Cs in rats following application of $^{137}CsCl$ solution to the skin. Although cesium was distributed throughout the body, it was deposited mainly in the kidneys, muscular tissues (particularly cardiac muscle), and liver.

3.4.2.4 Other Routes of Exposure

Comparative human and animal studies have shown that parenteral exposure to cesium compounds results in cesium distribution patterns similar to those observed following inhalation or oral exposure (Rosoff et al. 1963; Stara 1965). By 3 days post administration, ^{137}Cs was found to be distributed relatively uniformly among the body tissues of five cancer patients who died at various times following intravenous injection of a single tracer dose of ^{137}Cs (as cesium chloride). Time-related decreases were observed in ^{137}Cs retention by all tissues surveyed (Rosoff et al. 1963). Transfer of ^{137}Cs from pregnant dam to fetus has been shown in rats following intraperitoneal injection of radiolabeled cesium chloride (Mahlum and Sikov 1969).

3.4.3 Metabolism

Absorbed cesium behaves in a manner similar to that of potassium (Rundo 1964; Rundo et al. 1963). Both potassium and cesium are heavy alkali metals that distribute throughout the body as cations, and are incorporated into intracellular fluids by active transport mechanisms. Although the biokinetics of potassium and cesium may vary in affinity for various cell types removal rates, the similarities allow elimination rates for potassium to be used as an index of elimination rates for cesium (NRC 1983).

3.4.4 Elimination and Excretion

3.4.4.1 Inhalation Exposure

Urinary excretion is the major route of elimination of cesium. In dogs exposed to ^{137}Cs (as cesium chloride) by inhalation, rates of excretion of ^{137}Cs in the urine and feces were highest in the first 3 days postexposure, accounting for approximately 12 and 3% of the initial body burden, respectively. Urinary and fecal excretion of ^{137}Cs continued at lower rates through 130 days of analysis. Rates of elimination were determined by measuring the cesium remaining in the organs of rats sacrificed at 9 time points during 120 days post treatment. The elimination rates for specific tissues (muscle, kidney, liver, and lung) were similar to the whole-body rates, indicating that cesium did not selectively accumulate in any particular tissues (Boecker 1969b). Within 2.5 days following inhalation exposure to ^{137}Cs (as cesium chloride), guinea pigs had eliminated approximately 50% of the initial ^{137}Cs body burden in the urine and feces (Stara 1965). The urinary to fecal ratio for excretion was approximately 3:1 throughout 60 days of postexposure measurements, by which time, virtually all of the initial ^{137}Cs body burden had been eliminated. The urinary to fecal ratio for elimination of cesium in rats was about 3.5:1, with a biological half-time of elimination of approximately 4 days; 99% had been eliminated within 65 days postexposure (Stara and Thomas 1963).

No reports were located regarding routes of elimination and excretion of cesium in humans following inhalation exposure. However, kinetics of elimination of ^{137}Cs in two adult males accidently exposed to ^{137}Cs (as cesium sulfate) were studies by whole-body measurements of gamma emission. The measured biological half-times were 73 and 84 days (Miller 1964).

In dogs exposed by inhalation, elimination rates of ^{137}Cs from specific tissues were similar to the rate of whole-body elimination, indicating that ^{137}Cs did not selectively accumulate in certain tissues, but was

relatively uniformly eliminated from the body with a half-time of approximately 36–42 days (Boecker 1969b). Elimination rates of ^{137}Cs in guinea pigs and rats exposed by inhalation also did not vary significantly according to tissue type, although much shorter half-times of ^{137}Cs elimination (2.5 and 4 days) were observed for guinea pigs and rats, respectively (Stara 1965; Stara and Thomas 1963).

Relatively insoluble inhaled particles containing cesium were not absorbed in significant amounts and were more slowly eliminated from the lungs.

3.4.4.2 Oral Exposure

Urinary excretion is the primary route of elimination for cesium in humans. Among seven cancer or pulmonary patients who were administered single oral doses of ^{137}Cs (as cesium chloride), 7-day cumulative excretion of ^{137}Cs ranged from 7.0 to 17.3% of the administered activity. The urinary to fecal excretion ratio ranged from 2.5:1 to 10:1 (Rosoff et al. 1963). In a study of four Japanese volunteers orally administered single doses of ^{137}Cs (as cesium chloride), urinary to fecal excretion ratios ranging from 4.57:1 to 8.75:1 were calculated for excretory data collected after day 4 post administration. During the first 4 days post administration, excretory rates were consistently higher and the urinary:fecal excretory ratio was also somewhat higher (Iinuma et al. 1965). The principal sources of information on excretion rates of ^{137}Cs have been the numerous studies of populations exposed via fallout following atmospheric testing of nuclear weapons and from the Chernobyl accident, and mathematical models have been developed to describe the relationships among intake, retention, and elimination of cesium.

Animal data support the findings in humans of urinary excretion as the major route of elimination of cesium following oral administration of soluble cesium compounds. Guinea pigs had eliminated approximately 50% of the initial ^{137}Cs body burden in the urine and feces within 2.5 days postadministration (Stara 1965). The urinary to fecal ratio was within the range of 2 to 3:1 throughout 60 days of postexposure measurements, by which time, virtually all of the initial ^{137}Cs body burden had been eliminated.

Elimination half-times for cesium in the whole body, sometimes expressed in terms of whole-body radioactivity retention, have been reported by some investigators. Among 10 volunteers consuming ^{134}Cs and ^{137}Cs in a meal, approximately 94% of the absorbed radioactivity was more slowly eliminated following the rapid excretion of 6% of the initial body burden. The average half-times of the short- and long-term elimination components were 0.3 and 90 days, respectively (Henrichs et al. 1989). In another

oral study of four adult males, a mean elimination half-time was 135 days for ^{134}Cs and ^{137}Cs (Richmond et al. 1962).

Elimination rates for ^{137}Cs appear to be age- and sex-dependent, decreasing with age and lower in adult males than adult females. Studies of populations that consumed food containing ^{137}Cs from weapons testing fallout found the elimination half-time to vary from 15±5 days in infants to 100±50 days in adults (McCraw 1965). Similar studies after the Chernobyl accident found similar elimination half-times, which ranged from about 8 days for 1-year-old infants to about 110 days for adults (IAEA 1991). A 4-year study of 110 persons comprising a cross-section within an unspecified population indicated that children ages 5–14 had the shortest elimination half-times (20 days, with no significant difference between males and females). The elimination half-times in older groups were significantly longer (47 days for adolescent and adult females, 67 days in 15-year-old males, 93 days in males 30–50 years of age) (Boni 1969b). Melo et al. (1994) reported similar age- and sex-related differences in elimination rates among individuals internally contaminated by ^{137}Cs in the Goiânia, Brazil accident (see ATSDR 1999 for a complete description of the incident). In this case, a high correlation was found between biological half-time for ^{137}Cs and weight for all age groups and sexes, except adult females.

Elimination rates of cesium may be altered by potassium intake. Following the intraperitoneal injection of ^{137}Cs in rats, a basal diet supplemented with 8–11% potassium resulted in cesium clearance of 60 days compared to about 120 days for rats receiving the unsupplemented basal diet that contained 1% potassium (Richmond and Furchner 1961). After 20 days on the diets, rats receiving supplemental potassium had body burdens of ^{137}Cs that were one-half those of the rats not receiving supplemental potassium. This finding shows that supplemental potassium reduces the uptake and increases the elimination of ingested ^{137}Cs.

Cesium crosses the placenta from mother to fetus. Measurable amounts of ^{137}Cs have been detected in human placenta and fetal tissue (Toader et al. 1996; Yoshioka et al. 1976). Cesium concentrations are higher in older fetuses than in younger ones (Toader et al. 1996). Pregnancy may increase the removal of cesium from the mother, as indicated by shorter elimination half-times during pregnancy relative to measurements taken following pregnancy or among nonpregnant controls (Zundel et al. 1969). Retention of cesium was lower in males suffering from muscular dystrophy than in age-matched controls. Older males with advanced signs of muscular dystrophy had lower retention than younger males exhibiting earlier stages of the disease (Lloyd et al. 1973).

3.4.4.3 Dermal Exposure

No reports were located regarding elimination and excretion of cesium in humans or animals following dermal exposure.

3.4.4.4 Other Routes of Exposure

Evidence for age-related differences in cesium elimination rates was observed in rats injected with ^{134}Cs or ^{137}Cs (as cesium chloride) at various ages. Neonatal rats injected with cesium chloride exhibited higher retention than weaned or adult rats (Lengemann 1970; Mahlum and Sikov 1969). Retention in rats increased 14 times during the 1–4-month period of postnatal development; smaller increases were noted from 4 to 21 months with a slight decrease in cesium retention noted at 25 months of age (Lengemann 1970). Age-related increases in cesium retention rates were also observed in young dogs from 61 to approximately 300 days old, after which cesium retention reached a plateau; the increases in cesium retention were similar to growth curves (Tyler et al. 1969).

3.4.5 Physiologically Based Pharmacokinetic (PBPK) Pharmacodynamic (PD) Models

Physiologically based pharmacokinetic (PBPK) models use mathematical descriptions of the uptake and disposition of chemical substances to quantitatively describe the relationships among critical biological processes (Krishnan et al. 1994). PBPK models are also called biologically based tissue dosimetry models. PBPK models are increasingly used in risk assessments, primarily to predict the concentration of potentially toxic moieties of a chemical that will be delivered to any given target tissue following various combinations of route, dose level, and test species (Clewell and Andersen 1985). Physiologically based pharmacodynamic (PBPD) models use mathematical descriptions of the dose-response function to quantitatively describe the relationship between target tissue dose and toxic end points.

PBPK/PD models refine our understanding of complex quantitative dose behaviors by helping to delineate and characterize the relationships between: (1) the external/exposure concentration and target tissue dose of the toxic moiety, and (2) the target tissue dose and observed responses (Andersen and Krishnan 1994; Andersen et al. 1987). These models are biologically and mechanistically based and can be used to extrapolate the pharmacokinetic behavior of chemical substances from high to low dose, from route to route, between species, and between subpopulations within a species. The biological basis of

PBPK models results in more meaningful extrapolations than those generated with the more conventional use of uncertainty factors.

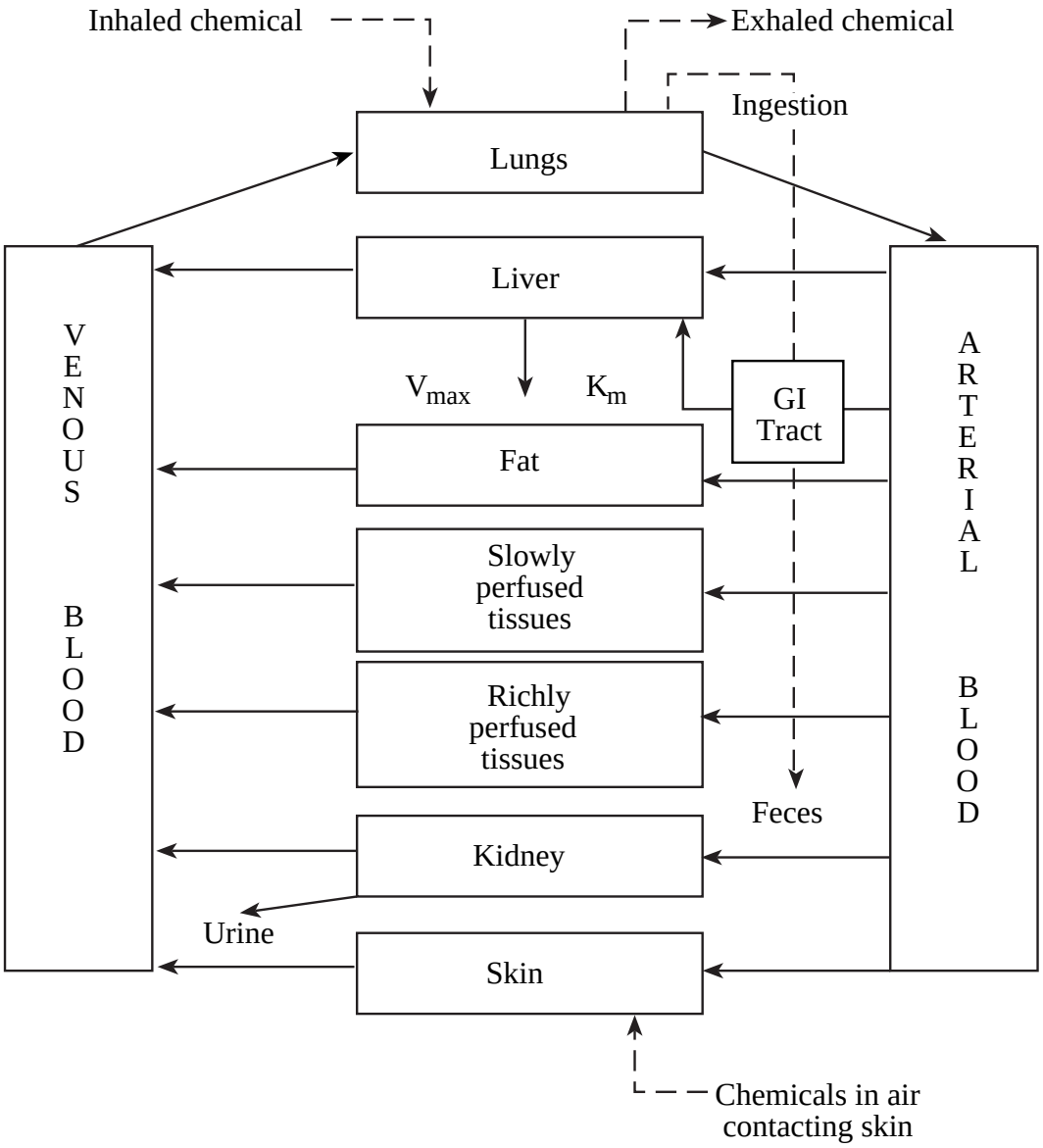
The PBPK model for a chemical substance is developed in four interconnected steps: (1) model representation, (2) model parametrization, (3) model simulation, and (4) model validation (Krishnan and Andersen 1994). In the early 1990s, validated PBPK models were developed for a number of toxicologically important chemical substances, both volatile and nonvolatile (Krishnan and Andersen 1994; Leung 1993). PBPK models for a particular substance require estimates of the chemical substance-specific physicochemical parameters, and species-specific physiological and biological parameters. The numerical estimates of these model parameters are incorporated within a set of differential and algebraic equations that describe the pharmacokinetic processes. Solving these differential and algebraic equations provides the predictions of tissue dose. Computers then provide process simulations based on these solutions.

The structure and mathematical expressions used in PBPK models significantly simplify the true complexities of biological systems. If the uptake and disposition of the chemical substance(s) is adequately described, however, this simplification is desirable because data are often unavailable for many biological processes. A simplified scheme reduces the magnitude of cumulative uncertainty. The adequacy of the model is, therefore, of great importance, and model validation is essential to the use of PBPK models in risk assessment.

PBPK models improve the pharmacokinetic extrapolations used in risk assessments that identify the maximal (i.e., the safe) levels for human exposure to chemical substances (Andersen and Krishnan 1994). PBPK models provide a scientifically sound means to predict the target tissue dose of chemicals in humans who are exposed to environmental levels (for example, levels that might occur at hazardous waste sites) based on the results of studies where doses were higher or were administered in different species. Figure 3-4 shows a conceptualized representation of a PBPK model.

The International Commission on Radiological Protection (ICRP 1994, 1995) developed a Human Respiratory Tract Model for Radiological Protection, which contains respiratory tract deposition and clearance compartmental models for inhalation exposure that may be applied to particulate aerosols of cesium compounds. The ICRP (1979, 1989, 1993) also developed a 2-compartment biokinetic model for human oral exposure that applies to cesium. EPA (1998) has adopted the ICRP (1993, 1994, 1995) models for assessment of radiologic risks from cesium exposures. The National Council on Radiation

Figure 3-4. Conceptual Representation of a Physiologically Based Pharmacokinetic (PBPK) Model for a Hypothetical Chemical Substance



Source: adapted from Krishnan et al. 1994

Note: This is a conceptual representation of a physiologically based pharmacokinetic (PBPK) model for a hypothetical chemical substance. The chemical substance is shown to be absorbed via the skin, by inhalation, or by ingestion, metabolized in the liver, and excreted in the urine or by exhalation.

Protection and Measurements (NCRP) has also developed a respiratory tract model for inhaled radionuclides (NCRP 1997). At this time, the NCRP recommends the use of the ICRP model for calculating radiation doses for workers and the general public. Readers interested in this topic are referred to NCRP Report No. 125; *Deposition, Retention and Dosimetry of Inhaled Radioactive Substances* (NCRP 1997). In the appendix to the report, NCRP provides the animal testing clearance data and equations fitting the data that supported the development of the human model for cesium.

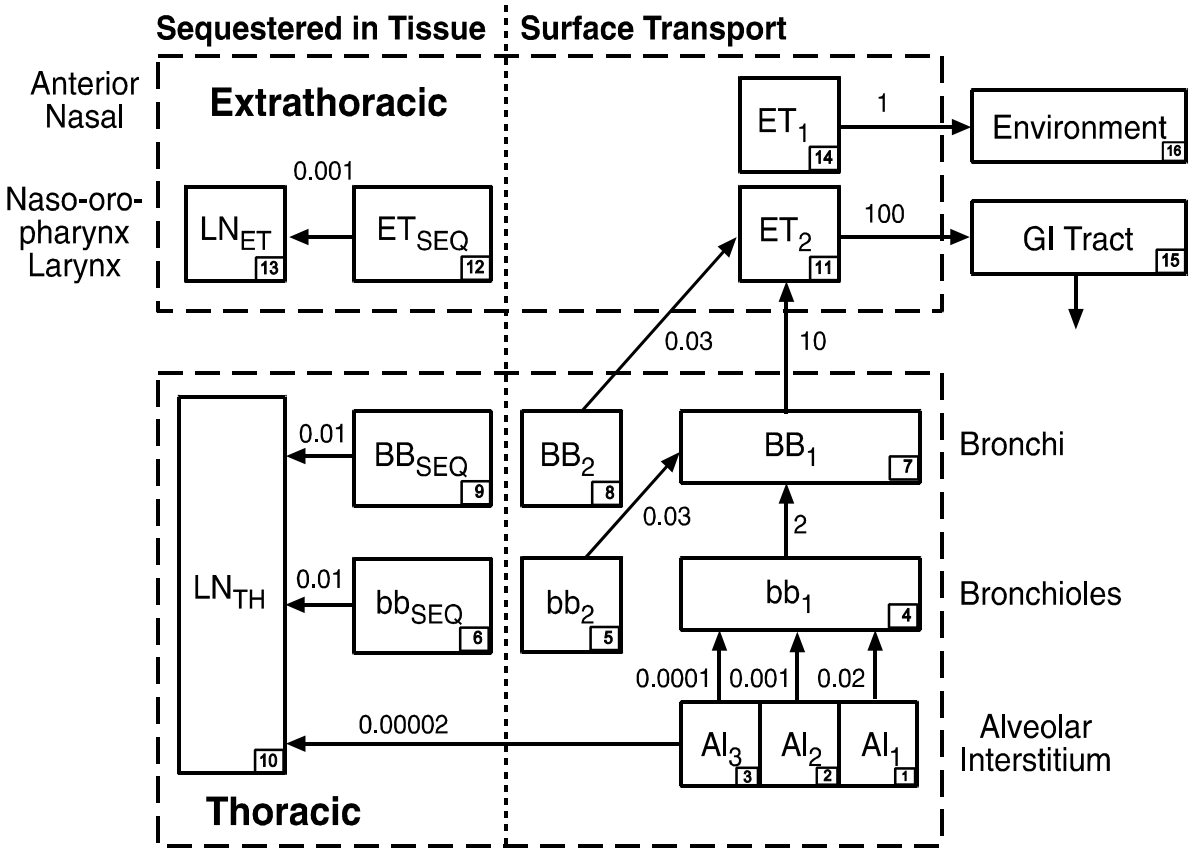
Human Respiratory Tract Model for Radiological Protection (ICRP 1994)

Deposition. The ICRP (1994) developed a deposition model to describe the behavior of inhaled aerosols and vapors in the respiratory tract. This model was developed to estimate the fractions of radioactivity in breathing air that are deposited in each anatomical region of the respiratory tract. ICRP (1994) provides inhalation dose coefficients, which can be used to estimate the committed equivalent and effective doses to organs and tissues throughout the body based on a unit intake of radioactive material. The model applies to three levels of particle solubility, a wide range of particle sizes (approximately 0.0005–100 μm in diameter), and parameter values. The model may be adjusted for various segments of the population (e.g., sex, age, level of physical exertion). This model also allows the evaluation of the bounds of uncertainty in deposition estimates. Uncertainties arise from natural biological variability among individuals. The ICRP model is applicable to particulate aerosols containing cesium, but was developed for a wide variety of radionuclides and their chemical forms.

The ICRP deposition model may be used to estimate the amount of inhaled material that initially enters each compartment (see Figure 3-5). The model was developed with 5 compartments: (1) the anterior nasal passages (ET_1); (2) all other extrathoracic airways (ET_2) (posterior nasal passages, the naso- and oropharynx, and the larynx); (3) the bronchi (BB); (4) the bronchioles (bb); and (5) the alveolar interstitium (AI). Particles deposited in the ET_1 region may be cleared either by dissolution into the blood or by nose-blowing. Particles deposited in each of the other regions may then be removed from each region and redistributed either upward into the respiratory tree by mucociliary clearance mechanisms, or to the lymphatic system and blood by different particle removal mechanisms.

For extrathoracic deposition of particles, the model is based on experimental data, where deposition is related to particle size and airflow parameters. The model scales deposition for women and children from adult male data. Similarly to the extrathoracic region, experimental data served as the basis for lung (bronchi, bronchioles, and alveoli) aerosol transport and deposition. A theoretical model of gas transport

Figure 3-5. Respiratory Tract Compartments in Which Particles May be Deposited



Size of arrow represents relative deposition rate

Source: ICRP 1994

and particle deposition was used to interpret data and to predict deposition for compartments and subpopulations other than adult males. Table 3-6 provides reference respiratory values for the general Caucasian population and for several levels of physical activity.

Deposition of inhaled gases and vapors is modeled as a partitioning process that depends on the physiological parameters noted above as well as the solubility and reactivity of compounds in the respiratory tract (see Figure 3-6). The ICRP (1994) model defines three categories of solubility and reactivity: SR-0, SR-1, and SR-2:

- Type SR-0 compounds include insoluble and nonreactive gases (e.g., inert gases such as H_2, He). These compounds do not significantly interact with the respiratory tract tissues and essentially all the inhaled gas is exhaled. Radiation doses from inhalation of SR-0 compounds are assumed to result from the irradiation of the respiratory tract from the air spaces.
- Type SR-1 compounds include soluble or reactive gases and vapors that are expected to be taken up by the respiratory tract tissues and may deposit in any or all of the regions of the respiratory tract, depending on the dynamics of the airways and properties of the surface mucous and airway tissues, as well as the solubility and reactivity of the compound.
- Type SR-2 compounds include soluble and reactive gases and vapors that are completely retained in the extrathoracic regions of the respiratory tract. SR-2 compounds include sulfur dioxide (SO_2) and hydrogen fluoride (HF).

Respiratory Tract Clearance. The clearance portion of the model identifies the principal clearance pathways within the respiratory tract. The model was developed to predict the retention and clearance of various radioactive materials. Figure 3-7 presents the compartmental model and is linked to the deposition model (Figure 3-5) and to reference values presented in Table 3-6. Table 3-7 provides clearance rates by biological processes only, not by radioactive decay, and deposition fractions for each compartment for insoluble particles. The table provides rates of insoluble particle transport for each of the compartments, expressed as a fraction per day and also as half-time. ICRP (1994) also developed modifying factors for some of the parameters, such as age, smoking and disease status. Parameters of the clearance model are based on human data, although particle retention in airway walls is based on experimental data from animal experiments.

The clearance of particles from the respiratory tract is a dynamic process. The rate of clearance generally changes with time from each region and by each route. Following deposition of large numbers of particles over a short time period (acute exposure), transport rates change as particles are cleared from the various regions. Physical and chemical properties of deposited material determine the rate of dissolution.

Table 3-6. Reference Respiratory Values for a General Caucasian Population at Different Levels of Activity[a]

Activity:		Resting (sleeping)			Sitting awake			Light exercise			Heavy exercise		
Maximal workload (%):		8			12			32			64		
Breathing parameters:[b]		V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})
Age	Sex												
3 months		0.04	0.09	38	N/A	N/A	N/A	0.07	0.19	48	N/A	N/A	N/A
1 year		0.07	0.15	34	0.1	0.22	36	0.13	0.35	46	N/A	N/A	N/A
5 years		0.17	0.24	23	0.21	0.32	25	0.24	0.57	39	N/A	N/A	N/A
10 years	Male:										0.841	2.22	44
	Both:	0.3	0.31	17	0.33	0.38	19	0.58	1.12	32			
	Female:										0.667	1.84	46
15 years	Male:	0.500	0.42	14	0.533	0.48	15	1.0	1.38	23	1.352	2.92	36
	Female:	0.417	0.35	14	0.417	0.40	16	0.903	1.30	24	1.127	2.57	38
Adult	Male:	0.625	0.45	12	0.750	0.54	12	1.25	1.5	20	1.923	3.0	26
	Female:	0.444	0.32	12	0.464	0.39	14	0.992	1.25	21	1.364	2.7	33

[a]See Annex B (ICRP 1994) for data from which these reference values were derived.
[b]B = ventilation rate; f_R = respiration frequency; V_T = tidal volume
h = hour; L = liter; m = meter; min = minute; N/A = not applicable

Table 3-7. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract

Part A

Clearance Rates for Insoluble Particles

Pathway	From	To	Rate (d^{-1})	Half-time[a]
$m_{1,4}$	AI_1	bb_1	0.02	35 days
$m_{2,4}$	AI_2	bb_1	0.001	700 days
$m_{3,4}$	AI_3	bb_1	0.0001	7,000 days
$m_{3,10}$	AI_3	LN_{TH}	0.00002	—
$m_{4,7}$	bb_1	BB_1	2	8 hours
$m_{5,7}$	bb_2	BB_1	0.03	23 days
$m_{6,10}$	bb_{seq}	LN_{TH}	0.01	70 days
$m_{7,11}$	BB_1	ET_2	10	100 minutes
$m_{8,11}$	BB_2	ET_2	0.03	23 days
$m_{9,10}$	BB_{seq}	LN_{TH}	0.01	70 days
$m_{11,15}$	ET_2	GI tract	100	10 minutes
$m_{12,13}$	ET_{seq}	LN_{ET}	0.001	700 days
$m_{14,16}$	ET_1	Environment	1	17 hours

See next page for Part B

Table 3-7. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract (*continued*)

Part B

Partition of deposit in each region between compartments[b]

Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment[c]
ET_2	ET_2	0.9995
	ET_{seq}	0.0005
BB	BB_1	0.993-f_s
	BB_2	f_s
	BB_{seq}	0.007
bb	bb_1	0.993-f_s
	bb_2	f_s
	bb_{seq}	0.007
AI	AI_1	0.3
	AI_2	0.6
	AI_3	0.1

[a]The half-times are approximate since the reference values are specified for the particle transport rates and are rounded in units of d^{-1}. A half-time is not given for the transport rate from AI_3 to LN_{TH}, since this rate was chosen to direct the required amount of material to the lymph nodes. The clearance half-time of compartment AI_3 is determined by the sum of the clearance rates from it.
[b]See paragraph 181, Chapter 5 (ICRP 1994) for default values used for relating f_s to d_{ae}.
[c]It is assumed that f_s is size-dependent. For modeling purposes, f_s is taken to be:

$$f_s = 0.5 \text{ for } d_{ae} \le 2.5\sqrt{\rho / \chi}\ \mu m \text{ and}$$

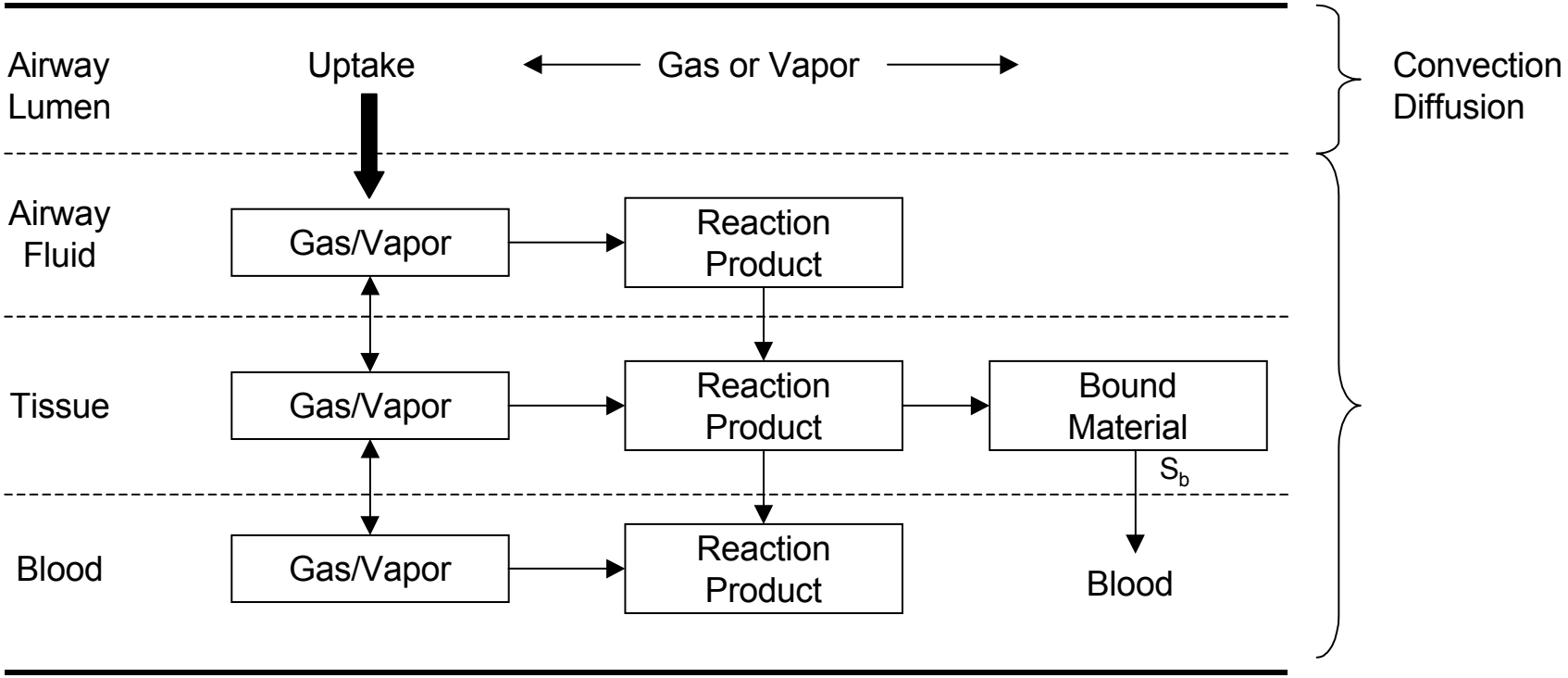
$$f_s = 0.5e^{0.63(d_{ae}\sqrt{\rho/\chi} - 2.5)} \text{ for } d_{ae} > 2.5\sqrt{\rho / \chi}\ \mu m$$

where
f_s = fraction subject to slow clearance
d_{ae} = aerodynamic particle diameter/(μm)
ρ = particle density (g/cm^3)
χ = particle shape factor

AI = alveolar-interstitial region; BB = bronchial region; bb = bronchiolar region; BB_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchial region; bb_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchiolar region; d = day(s); ET = extrathoracic region; ET_{seq} = compartment representing prolonged retention in airway tissue of small fraction of particles deposited in the nasal passages; LN_{ET} = lymphatics and lymph nodes that drain the extrathoracic region; LN_{TH} = lymphatics and lymph nodes that drain the thoracic region

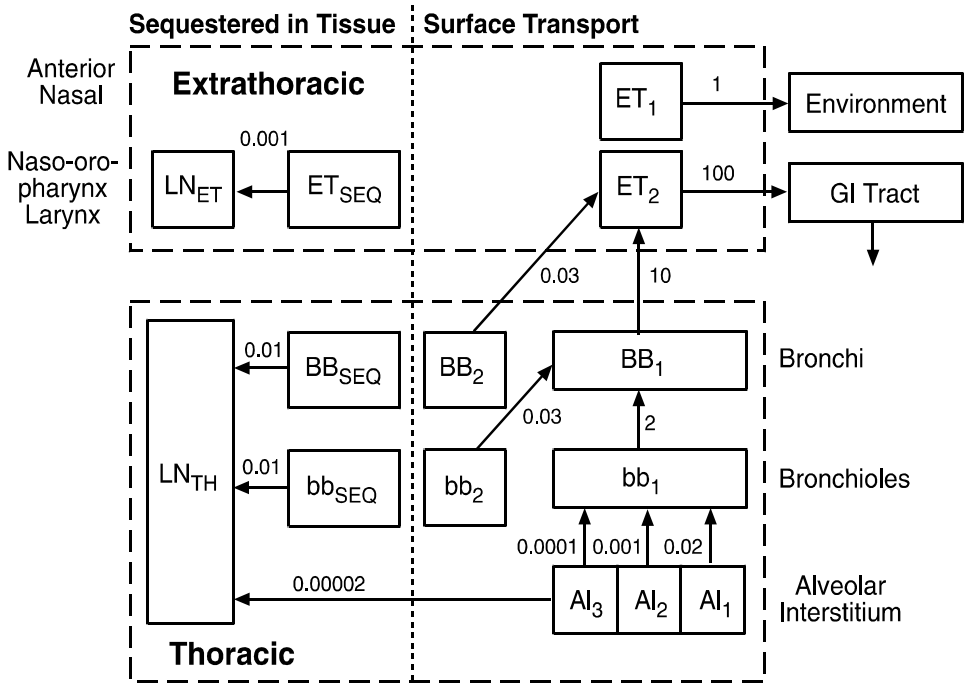
Source: ICRP 1994

Figure 3-6. Reaction of Gases or Vapors at Various Levels of the Gas-Blood Interface



From ICRP (1994)

Figure 3-7. Compartment Model to Represent Time-Dependent Particle Transport in the Respiratory Tract



Source: ICRP 1994

(See Table 3-7 for rates, half-lives, and fractions by compartment)

As particles dissolve, absorption rates also tend to change over time. By creating a model with compartments of different clearance rates within each region (e.g., BB_1, BB_2, BB_{seq}), the ICRP model overcomes problems associated with time-dependent functions. Each compartment clears to other compartments by constant rates for each pathway.

Particle transport from all regions is toward both the lymph nodes and the pharynx. A majority of deposited particles are eventually swallowed. In the front part of the nasal passages (ET_1), nose blowing, sneezing, and wiping remove most of the deposited particles. Particles remain in the nasal passages for about a day. For particles with AMADs of a few micrometers or greater, the ET_1 compartment is probably the largest deposition site. A majority of particles deposited at the back of the nasal passages and in the larynx (ET_2) are removed quickly by the mucous fluids that cover the airways. In this region, particle clearance is completed within 15 minutes.

Ciliary action removes deposited particles from the bronchi and bronchioles. Though mucociliary action rapidly transports most particles deposited here toward the pharynx, a fraction of these particles is cleared more slowly. Evidence for this clearance is found in human studies. For humans, retention of particles deposited in the lungs (BB and bb) is apparently biphasic. The "slow" action of the cilia may remove as many as half of the bronchi- and bronchiole-deposited particles. In human bronchi and bronchiole regions, mucus movement is influenced by location. Movement is slower in areas closer to alveoli. It takes about 2 days for particles to travel from the bronchioles to the bronchi and 10 days from the bronchi to the pharynx. The second (slower) compartment is assumed to have approximately equal fractions deposited between BB_2 and bb_2 and both with clearance half-times estimated at 20 days. Particle size is a primary determinant of the fraction deposited in this slow thoracic compartment. A small fraction of particles deposited in the BB and bb regions may be retained in the airway wall for even longer periods (BB_{seq} and bb_{seq}).

If particles reach and become deposited in the alveoli, they tend to stay imbedded in the fluid on the alveolar surface or move into the lymph nodes. The mechanism by which particles are physically resuspended and removed from the AI region is coughing. For modeling purposes, the AI region is divided into 3 subcompartments representing different slow clearance rates, all of which are slow.

Human lung clearance has been measured in the alveolar-interstitial region. The ICRP model uses 2 half-times to represent clearance. About 30% of the particles have a 30-day half-time, and the remaining 70%

are given a half-time of several hundred days. Over time, AI particle transport falls and some insoluble particles may remain in lungs 10–50 years after exposure.

Absorption into Blood. The ICRP model assumes that absorption into blood occurs at equivalent rates in all parts of the respiratory tract, except in the anterior nasal passages (ET_1), where no absorption occurs. Absorption is essentially a 2-stage process, as shown in Figure 3-8. First, there is a dissociation (dissolution) of particles. The dissolved molecules or ions then diffuse across capillary walls and are taken up by the blood. Immediately following dissolution, rapid absorption takes place. For some elements, rapid absorption does not occur because of binding to respiratory-tract components. In the absence of specific data for specific compounds, the model uses the following default absorption rate values for those specific compounds that are classified as Types F (fast), M (medium), S (slow), and V (instantaneous):

- For Type F, there is rapid 100% absorption within 10 minutes of the material deposited in the BB, bb, and AI regions, and 50% of material deposited in ET_2. Thus, for nose breathing, there is rapid absorption of approximately 25% of the deposit in ET and 50% for mouth breathing.

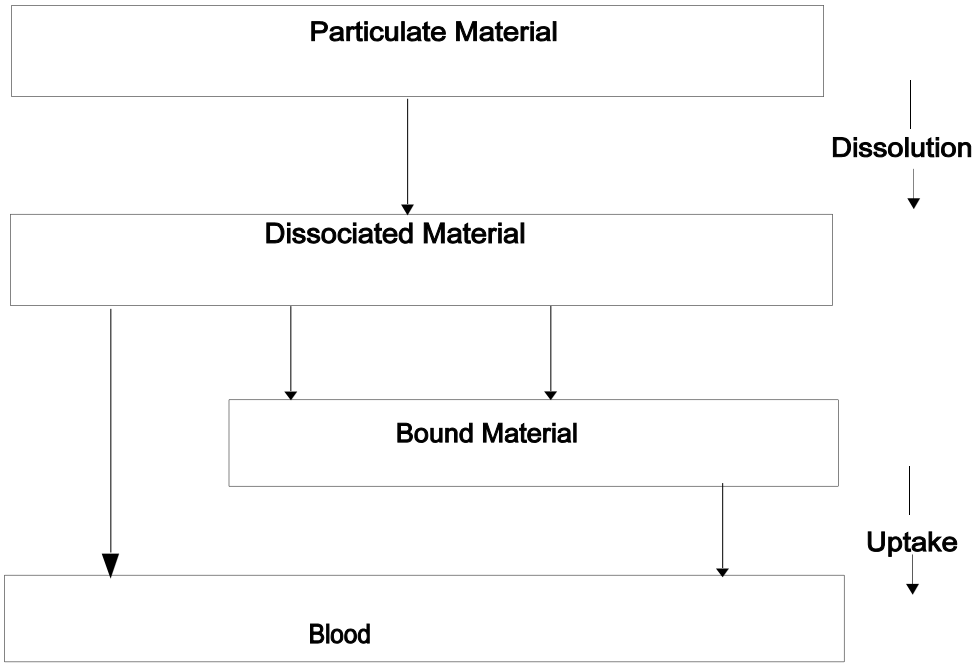
- For Type M, about 70% of the deposit in AI reaches the blood eventually. There is rapid absorption of about 10% of the deposit in BB and bb, and 5% of material deposited in ET_2. Thus, there is rapid absorption of approximately 2.5% of the deposit in ET for nose breathing, and 5% for mouth breathing.

- For Type S, 0.1% is absorbed within 10 minutes and 99.9% is absorbed within 7,000 days, so there is little absorption from ET, BB, or bb, and about 10% of the deposit in AI reaches the blood eventually.

- For Type V, complete absorption (100%) is considered to occur instantaneously.

ICRP (1995) considers the experimental and human data to support the following classifications: cesium chloride and nitrate, Type F; cesium in irradiated fuel fragments, Type F or M; cesium in fused aluminosilicate particles, M or S. ICRP (1995) recommends assigning all cesium aerosols to Type F in the absence of specific information supporting an alternative classification.

Figure 3-8. The Human Respiratory Tract Model: Absorption into Blood



Source: ICRP 1994

ICRP (1993) Cesium Biokinetic Model

Description of the model.

ICRP (1979, 1989, 1993) developed a 2-compartment model of the kinetics of ingested cesium in humans that is applicable to infants, children, adolescents, and adults. The model is based on similar models described for potassium and cesium (NRC 1983). Ingested cesium is assumed to be completely absorbed into blood. Absorbed cesium is then assumed to distribute uniformly in the body and to be eliminated from fast and slow elimination pools. The fraction of the total body cesium associated with the fast and slow pools, as well as the elimination half-times from each pool, are assumed to vary with age. The elimination half-times vary for ages 3 months, 1, 5, 10, 15 years, and adult (>15 years). The contribution of the fast pool decreases from 45% at age 5 years to 10% in adults. The elimination half-times of the fast pool decrease from 9.1 days at age 5 years to 2 days in adults, whereas the elimination half-times of the slow pool increase from 13 to 16 days in infants to 110 days in adults.

Validation of the model.

The extent to which the ICRP model has been validated is not described in ICRP (1993).

Risk assessment.

The ICRP biokinetic model has been used to establish radiation dose equivalents (Sv/Bq) of ingested ^{134}Cs, ^{136}Cs, and ^{137}Cs for ages 3 months to 70 years (ICRP 1993).

Target tissues.

The ICRP model is designed to calculate radiocesium intake limits, based on radiation dose to all major organs.

Species extrapolation.

The ICRP model is designed for applications to human dosimetry and cannot be applied to other species without modification.

Interroute extrapolation.

The ICRP model is designed to simulate oral exposures to cesium and cannot be applied to other routes of exposure without modification.

3.5 MECHANISMS OF ACTION

3.5.1 Pharmacokinetic Mechanisms

Cesium is rapidly absorbed into blood following inhalation or oral exposure to soluble cesium compounds, as demonstrated by the rapid distribution of cesium activity after inhalation or ingestion. Approximately 80% absorption of ^{137}Cs was observed in dogs exposed to aerosols containing ^{137}Cs (as cesium chloride) (Boecker 1969a, 1969b). Oral ingestion of ^{134}Cs- and ^{137}Cs-contaminated food by volunteers resulted in approximately 78% absorption. Animal studies indicate that absorption rates from orally-administered soluble cesium compounds are highest in the duodenum, followed in order by the jejunum, ileum, and colon. Very little absorption occurs in the stomach or caecum (Moore and Comar 1962, 1963). Absorption rates are higher in fasted rats than in fed rats, indicating that stomach contents may influence the rate of cesium absorption. Relatively insoluble forms of cesium compounds, which may sometimes be associated with irradiated fuel particles, are poorly absorbed by inhalation and oral exposure routes (Boecker et al. 1974, 1977; LeRoy et al. 1966; Talbot et al. 1993). Dermal retention, but not transdermal absorption, has been qualitatively demonstrated in humans (Rundo 1964). Dermal absorption was observed following application of ^{137}CsCl in aqueous solution to the skin of rats (Pendic and Milivojevic 1966). Traces of ^{137}Cs were observed in the blood of rats within a few minutes following application.

Once absorbed, cesium is rapidly distributed throughout the body, becoming incorporated into the intracellular fluid of numerous tissues. Animal studies indicate that distribution patterns are similar following absorption from inhalation or oral exposure and that concentrations of cesium within muscle tissue are somewhat higher than the whole-body average (Stara 1965). Comparative human and animal studies have shown that parenteral exposure to cesium compounds results in cesium distribution patterns similar to those following inhalation or oral exposure (Rosoff et al. 1963; Stara 1965).

Absorbed cesium behaves in a manner similar to that of potassium. Both potassium and cesium are heavy alkali metals that are distributed throughout the body as cations, becoming incorporated into intracellular

fluids via active transport mechanisms. Although the biokinetics of potassium and cesium may vary somewhat in such characteristics as relative affinity for various cell types and differing retention rates, the similarities allow elimination rates for potassium to be used as an index of elimination rates for cesium (NRC 1983). Urinary excretion is the major route of elimination of cesium.

3.5.2 Mechanisms of Toxicity

Stable Cesium. Earth contains relatively small amounts of stable (nonradioactive) cesium. Cesium has few industrial applications. At environmental levels, stable cesium is not chemically toxic in animals. Cesium is not likely to be of toxic concern to humans exposed to cesium by inhalation, oral, or dermal contact. Although a number of investigators have reported cesium-induced alterations in behavior or cardiac activity in animals systems exposed to cesium chloride by parenteral injection, underlying mechanisms are not yet fully understood.

Cesium may have both depressant and antidepressant properties in rodents, as it was shown to decrease the conditioned avoidance response of pole-climbing (Bose and Pinsky 1983b) and to reduce vertical and horizontal motor activity (Bose and Pinsky 1981, 1984; Bose et al. 1981; Pinsky et al. 1980), while enhancing amphetamine-induced hyperactivity and reducing the locomotor depressive action of reserpine (Messiha 1978).

Increased vertical activity (rearing), but not horizontal activity, was observed in mice given repeated injections of cesium chloride (Johnson 1972). Rastogi et al. (1980) found no increase in behavioral activity in rats repeatedly injected with cesium chloride, but noted a number of biochemical changes in the brain that included a statistically significant rise in tyrosine hydroxylase activity that resulted in a slight but statistically significant increase in tyrosine levels, markedly enhanced levels of the neurotransmitters norepinephrine and dopamine, and increased levels of a norepinephrine metabolite (4-hydroxy-3-methoxyphenylglycol). Cesium appeared to block the uptake of norepinephrine by synaptosomes.

Cesium was shown to alter normal cardiac rhythm, triggering short-lived early after depolarizations (EADs) and polymorphic ventricular tachyarrhythmias (VTs) in canine myocardial muscle fibers and Purkinje cells (Brachmann et al. 1983; Levine et al. 1985; Murakawa et al. 1997; Patterson et al. 1990), effects that are similar to those observed in humans with congenital and acquired long Q-T syndrome (Bonatti et al. 1983). Although the mechanisms responsible for these effects have not been elucidated,

available animal data suggest that cesium-induced EADs and VTs were most likely the result of ionic imbalance due to reduced potassium permeability (Isenberg 1976) and imbalances of intra- and extracellular concentrations of calcium and sodium (Szabo et al. 1987).

Radioactive Cesium. Both ^{134}Cs and ^{137}Cs emit gamma radiation, and therefore radioactive cesium may be a health hazard. Highly-penetrating gamma rays are the major cause of damage to tissues and internal organs following external overexposure to radioactive cesium. Once radioactive cesium is taken internally, cells of nearby tissues are at highest risk for damage due to the emission of beta particles. Radiation-induced damage in cells may be repaired quickly. Misrepaired damage may lead to permanent DNA changes and the potential for carcinogenesis. Very large acute radiation doses can damage or kill enough cells to cause the disruption of organ systems (acute radiation syndrome), harm to developing fetuses, and even death. Human and animal data indicate that radioactive cesium overexposure can result in adverse effects such as reduced fertility, abnormal neurological development, genotoxicity, and damage to blood-forming organs (Bartstra et al. 1998; Matsuda et al. 1985; Nikula et al. 1995, 1996; Padovani et al. 1993; Ramaiya et al. 1994; Skandalis et al. 1997; Tobari et al. 1988). For a more complete discussion of the mechanisms associated with the toxic effects of ionizing radiation, refer to Chapter 5 of the Toxicological Profile for Ionizing Radiation (ATSDR 1999).

3.5.3 Animal-to-Human Extrapolations

No data were located to indicate significant interspecies differences in pharmacokinetics or health effects associated with exposure to stable or radioactive cesium.

3.6 ENDOCRINE DISRUPTION

Recently, attention has focused on the potential hazardous effects of certain chemicals on the endocrine system because of the ability of these chemicals to mimic or block endogenous hormones, or otherwise interfere with the normal function of the endocrine system. Chemicals with this type of activity are most commonly referred to as endocrine disruptors. Some scientists believe that chemicals with the ability to disrupt the endocrine system are a potential threat to the health of humans, aquatic animals, and wildlife. Others believe that endocrine disrupting chemicals do not pose a significant health risk, particularly in light of the fact that hormone mimics exist in the natural environment. Examples of natural hormone mimics are the isoflavinoid phytoestrogens (Adlercreutz 1995; Livingston 1978; Mayr et al. 1992). These compounds are derived from plants and are similar in structure and action as endogenous estrogen. While

there is some controversy over the public health significance of endocrine disrupting chemicals, it is agreed that the potential exists for these compounds to affect the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, reproduction, development, and/or behavior (EPA 1997). As a result, endocrine disruptors may play a role in the disruption of sexual function, immune suppression, and neurobehavioral function. Endocrine disruption is also thought to be involved in the induction of breast, testicular, and prostate cancers, as well as endometriosis (Berger 1994; Giwercman et al. 1993; Hoel et al. 1992).

No studies were located regarding endocrine disruptive effects resulting from exposure to stable or radioactive cesium.

3.7 CHILDREN'S SUSCEPTIBILITY

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans, when all biological systems will have fully developed. Potential effects on offspring resulting from exposures of parental germ cells are considered, as well as any indirect effects on the fetus and neonate resulting from maternal exposure during gestation and lactation. Relevant animal and *in vitro* models are also discussed.

Children are not small adults. They differ from adults in their exposures and may differ in their susceptibility to hazardous chemicals. Children's unique physiology and behavior can influence the extent of their exposure. Exposures of children are discussed in Section 6.6 Exposures of Children.

Children sometimes differ from adults in their susceptibility to hazardous chemicals, but whether there is a difference depends on the chemical (Guzelian et al. 1992; NRC 1993a). Children may be more or less susceptible than adults to health effects, and the relationship may change with developmental age (Guzelian et al. 1992; NRC 1993a). Vulnerability often depends on developmental stage. There are critical periods of structural and functional development during both prenatal and postnatal life and a particular structure or function will be most sensitive to disruption during its critical period(s). Damage may not be evident until a later stage of development. There are often differences in pharmacokinetics and metabolism between children and adults. For example, absorption may be different in neonates because of the immaturity of their gastrointestinal tract and their larger skin surface area in proportion to body weight (Morselli et al. 1980; NRC 1993a); the gastrointestinal absorption of lead is greatest in infants and young children (Ziegler et al. 1978). Distribution of xenobiotics may be different; for

example, infants have a larger proportion of their bodies as extracellular water and their brains and livers are proportionately larger (Altman and Dittmer 1974; Fomon 1966; Fomon et al. 1982; Owen and Brozek 1966; Widdowson and Dickerson 1964). The infant also has an immature blood-brain barrier (Adinolfi 1985; Johanson 1980) and probably an immature blood-testis barrier (Setchell and Waites 1975). Many xenobiotic metabolizing enzymes have distinctive developmental patterns. At various stages of growth and development, levels of particular enzymes may be higher or lower than those of adults, and sometimes unique enzymes may exist at particular developmental stages (Komori et al. 1990; Leeder and Kearns 1997; NRC 1993a; Vieira et al. 1996). Whether differences in xenobiotic metabolism make the child more or less susceptible also depends on whether the relevant enzymes are involved in activation of the parent compound to its toxic form or in detoxification. There may also be differences in excretion, particularly in newborns who all have a low glomerular filtration rate and have not developed efficient tubular secretion and resorption capacities (Altman and Dittmer 1974; NRC 1993a; West et al. 1948). Children and adults may differ in their capacity to repair damage from chemical insults. Children also have a longer remaining lifetime in which to express damage from chemicals; this potential is particularly relevant to cancer.

Certain characteristics of the developing human may increase exposure or susceptibility, whereas others may decrease susceptibility to the same chemical. For example, although infants breathe more air per kilogram of body weight than adults breathe, this difference might be somewhat counterbalanced by their alveoli being less developed, which results in a disproportionately smaller surface area for alveolar absorption (NRC 1993a).

Soluble cesium compounds are readily absorbed into body fluids and bloodstream and are widely distributed throughout the body (see Section 3.4 for detailed information). PBPK models are used to simulate potential age-related differences in deposition of inhaled cesium, as well as differences in elimination rates for absorbed cesium (see Section 3.4.5 for more information on PBPK models). Although inhalation exposure to environmental levels of stable or radioactive cesium is not considered to be a major health concern, age-related differences in physical properties of the respiratory system and ventilation patterns could result in differences in absorption rates of inhaled soluble or insoluble cesium compounds. Soluble cesium compounds are assumed to be completely absorbed in the gastrointestinal tract, with no adjustments for age. Available human and animal data do not indicate that age-related differences might exist for absorption and distribution of cesium following oral exposure. Since cesium is principally absorbed and distributed in ionic form (as Cs^+), any age-related differences in absorption following oral exposure would likely be the result of differences in diffusion rates and/or active transport

mechanisms involved in the movement of cesium through extra- and intracellular fluids. Elimination rates for cesium appear to be age-related and may be most closely related to body mass. As described in detail in Section 3.4.4.2, young children exhibit whole-body biological elimination half-times that are much shorter than those of older children and adults (Boni 1969b; Melo et al. 1994). It is not known whether or not these age-related differences may be due to higher retention of cesium in adult tissues and lower rates of excretion.

Measurable amounts of ^{137}Cs have been found in the breast milk of women living in areas contaminated with radioactive fallout. Transfer to newborns and 1-year-old children was estimated to be approximately 40 and 50%, respectively (Johansson et al. 1998). Animal studies have shown that cesium crosses the placental barrier, but cesium is found in lower concentrations in the fetus than in maternal or placental tissues (Mahlum and Sikov 1969; Vandecasteele et al. 1989).

Health Effects from Exposure to Stable Cesium. An investigator who voluntarily ingested about 68 mg/kg/day of stable cesium (as cesium chloride) for 36 days reported symptoms of decreased appetite, nausea, diarrhea, and short-term feelings of well-being, heightened sense perception, and tingling sensations (Neulieb 1984). Although no information was located regarding health effects in children exposed to stable cesium, age-related differences in the pharmacokinetics of stable cesium could conceivably result in age-related differences in health effects. No studies were located regarding age-related differences in toxicity in animals exposed to stable cesium.

Health Effects from Exposure to Radioactive Cesium. Most of the available information regarding age-related health effects from overexposure to cesium concern developmental effects related to *in utero* irradiation of human or animal fetuses from an external source of radiation. Impaired cognitive function was observed in atomic bomb survivors overexposed to ionizing radiation *in utero* during critical stages of neurological development (Schull and Otake 1999).

Developmental toxicity studies employing external gamma radiation from a radioactive cesium source (or from any other gamma ray source) indicate that rats and mice are most sensitive to the effects of external radiation around gestation day 14. Effects observed following irradiation during this period include reduced survival, decreased brain size, smaller head size, and retarded odontogenesis (Koshimoto et al. 1994; Minamisawa et al. 1990; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994). When tested as adults, animals irradiated during this developmental period exhibit increased aggressive behavior (Minamisawa et al. 1992; Norton and Kimler 1987, 1988). Although comparative studies of neurological

effects in animals first irradiated as juveniles or adults were not located, it is apparent that there are critical stages of fetal developmental during which there is increased susceptibility to the effects of radiation. In these studies, although cesium was used as the gamma source, the effects were not unique to cesium. Similar results would be elicited by any gamma source.

3.8 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

Due to a nascent understanding of the use and interpretation of biomarkers, implementation of biomarkers as tools of exposure in the general population is very limited. A biomarker of exposure is a xenobiotic substance or its metabolite(s) or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s), or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time samples can be taken. It may be difficult to identify individuals exposed to hazardous substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as copper, zinc, and selenium). Biomarkers of exposure to cesium are discussed in Section 3.8.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are not often substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by cesium are discussed in Section 3.8.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, a decrease in the biologically effective dose, or a target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 3.10 "Populations That Are Unusually Susceptible".

3.8.1 Biomarkers Used to Identify or Quantify Exposure to Cesium

The biomarkers that may help quantify exposure to stable or radioactive cesium are similar in children and adults.

Stable or radioactive isotopes of cesium may be measured in samples of urine, blood, feces, or body tissues by a number of methods outlined in Section 7.1. Stable cesium is of little toxicological concern. However, overexposure to radioactive isotopes of cesium may pose a significant health risk. Internal exposure may be quantified by direct counting (*in vivo* measurements) of radioactive emission from the body using whole-body counters capable of distinguishing emissions that are unique to radioactive isotopes of cesium. Radioactivity can be accurately measured in blood, excrement, and tissue samples using scintillation counting.

3.8.2 Biomarkers Used to Characterize Effects Caused by Cesium

There are no known biomarkers of effect for exposure to stable cesium. High-level external or internal exposure to radioactive cesium can result in bone marrow aplasia, reduced white blood cell counts, decreased hemoglobin and platelet levels, and increased frequencies of chromosomal aberrations in lymphocytes. Frequencies of chromosomal aberrations were used to estimate external radiation doses among individuals in Goiânia, Brazil who had been exposed to a ^{137}Cs source (Natarajan et al. 1998). These results are not unique to radioactive cesium. Similar results would be expected following overexposure to any source of ionizing radiation.

3.9 INTERACTIONS WITH OTHER CHEMICALS

No data were located regarding interactions of cesium with other chemicals that might influence the toxicity of cesium.

3.10 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to cesium than will most persons exposed to the same level of cesium in the environment. Reasons may include genetic makeup, age, health and nutritional status, and exposure to other toxic substances (e.g., cigarette smoke). These parameters result in reduced detoxification or excretion of cesium, or compromised function of organs affected by cesium. Populations who are at greater risk due to their unusually high exposure to cesium are discussed in Section 6.7, Populations With Potentially High Exposures.

Increased susceptibility to the toxic effects resulting from exposure to high levels of stable or radioactive cesium might be indicated among individuals with abnormally low potassium intake, those with compromised kidney function, and patients taking stimulant or depressant drugs for the treatment of mental disorders (see Sections 3.4 and 3.5 for detailed information on the toxicokinetics and mechanisms of action of cesium). Individuals with compromised immune function might be more susceptible to the adverse effects of radiation overexposure from a radioactive cesium source (or from any other gamma emitting radioactive source).

Evidence for potential age-related differences in susceptibility to stable or radioactive cesium toxicity is provided by studies of elimination rates for ^{137}Cs in humans (Boni 1969b; Melo et al. 1994; Toader et al. 1996). Elimination rates are higher in young children than adults, and higher in adult females than adult males, indicating that lower elimination rates could result in greater retention and, therefore, increased toxicity for a given intake. Animal studies support these findings (Lengemann 1970; Mahlum and Sikov 1969; Melo et al. 1996, 1997; Tyler et al. 1969). There is some indication, however, that retention of cesium is higher in neonatal rats than in young adult rats (Mahlum and Sikov 1969) (see Sections 3.4.4.2 and 3.4.4.4 for more detailed information). Higher cesium retention was also shown in puppies (Melo et al. 1996).

Pregnant women (Zundel et al. 1969) and individuals suffering from muscular dystrophy (Lloyd et al. 1973) exhibited decreased cesium retention, which may decrease susceptibility to stable or radioactive cesium-induced toxicity.

3.11 METHODS FOR REDUCING TOXIC EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to cesium. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to cesium. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice. The following texts provide specific information about treatment following exposures to cesium:

Ellenhorn MJ, Schonwald S, Ordog G, et al., eds. 1997. Medical toxicology: Diagnosis and treatment of human poisoning. 2nd edition. Baltimore, MD: Williams & Wilkins. 1682-1723.

Haddad LM, Shannon MW, Winchester JF, eds. 1998. Clinical management of poisoning and drug overdose. 3rd edition. Philadelphia, PA: WB Saunders. 413-425.

NCRP Report No. 65. 1980. Management of persons accidently contaminated with radionuclides. Bethesda MD: National Council on Radiation Protection and Measurements.

3.11.1 Reducing Peak Absorption Following Exposure

Because soluble cesium compounds are rapidly absorbed into blood following inhalation, oral, and dermal exposure, there are no prescribed methods for reducing peak absorption following exposure. Early counter measures that could possibly aid in reducing peak absorption following oral exposure to stable or radioactive cesium include oral administration of Prussian blue (potassium ferricyanoferrate) that exchanges potassium for cesium, forming an insoluble complex that is eliminated through the feces. Cathartics such as magnesium sulfate, as well as gastric lavage, will shorten the transit time of ingested cesium in the gastrointestinal tract (Ellenhorn et al. 1997; Gerber et al. 1992; Haddad et al. 1998). Counter measures attempt to reduce the body burden of cesium following inadvertent exposure (see Section 3.11.2).

3.11.2 Reducing Body Burden

Oral administration of Prussian blue (potassium ferricyanoferrate) may enhance the fecal excretion of absorbed cesium (Ducousso et al. 1975; Ellenhorn et al. 1997; Gerber et al. 1992; Haddad et al. 1998; Melo et al. 1996). Animal studies indicate that excretion of cesium may also be enhanced by the administration of potassium-supplemented diets (Richmond and Furchner 1961). Higher plasma concentrations of potassium may increase the mobilization of cesium from tissues, and thus increase excretion.

3.11.3 Interfering with the Mechanism of Action for Toxic Effects

No data were located regarding reduction of the toxic effects of cesium through interfering with mechanisms of action.

3.12 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cesium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cesium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

3.12.1 Existing Information on Health Effects of Cesium

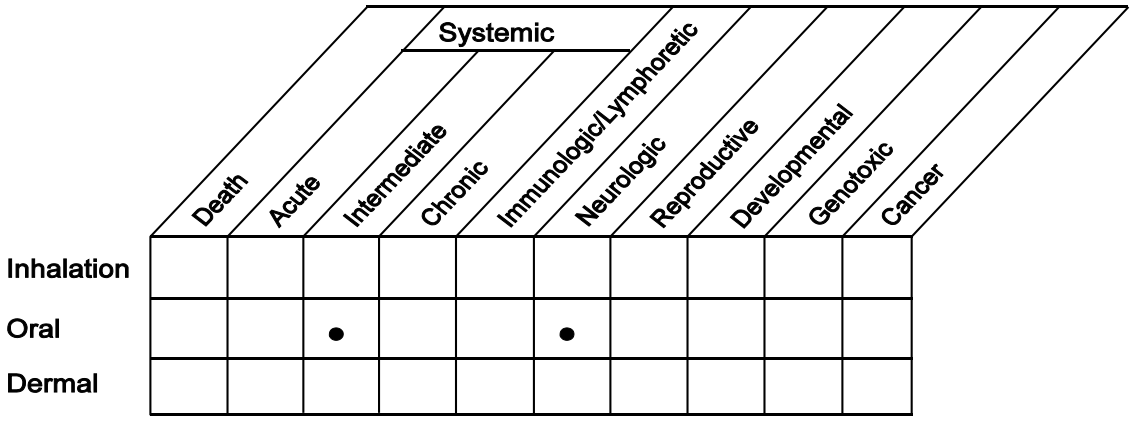
The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to cesium are summarized in Figure 3-9 for stable cesium and Figure 3-10 for radioactive cesium. The purpose of these figures is to illustrate the existing information concerning the health effects of cesium. Each dot in the figures indicates that one or more studies provide information associated with that particular effect. The dot does not necessarily imply anything about the quality of the study or studies, nor should missing information in these figures be interpreted as a "data need". A data need, as defined in ATSDR's *Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles* (ATSDR 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

Stable Cesium. As shown in Figure 3-9, limited information is available regarding health effects in humans following intermediate-duration oral exposure to stable cesium. No information is available regarding health effects in humans following inhalation or dermal exposure to stable cesium. Information is available on the health effects in animals exposed to stable cesium. However, the available information is mostly from acute oral LD_{50} studies, a single intermediate-duration oral study, and a study of male reproductive toxicity.

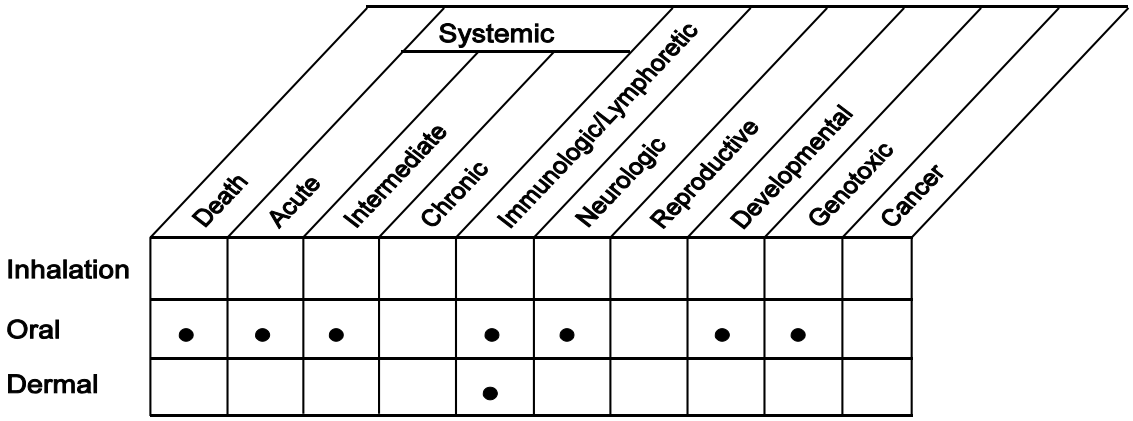
Radioactive Cesium. As shown in Figure 3-10, limited information is available regarding oral and dermal exposure to radioactive cesium. An accidental exposure of a number of individuals in Goiânia, Brazil resulted in adverse health effects that could be attributed to external and internal (oral and dermal) exposure to radiation from a radioactive cesium source. Numerous reports are available regarding other cases of external and internal environmental exposure to radioactive isotopes of cesium in humans, especially from areas with significant amounts of radioactive fallout. However, at present, associations between exposure to environmental levels of radioactive cesium and adverse health effects have not been confirmed. Present environmental levels of radiocesium, therefore, might not represent overexposure to radiation.

Reduced sperm counts in mice were found following oral administration of radioactive cesium. Studies of dogs, intravenously administered $^{137}CsCl$, resulted in hematologic dyscrasia as early effects and tumors of various organs as late effects. Given by this route, the tissue distribution of ^{137}Cs is similar to that resulting from oral or inhalation exposure.

Figure 3-9. Existing Information on Health Effects of Stable Cesium



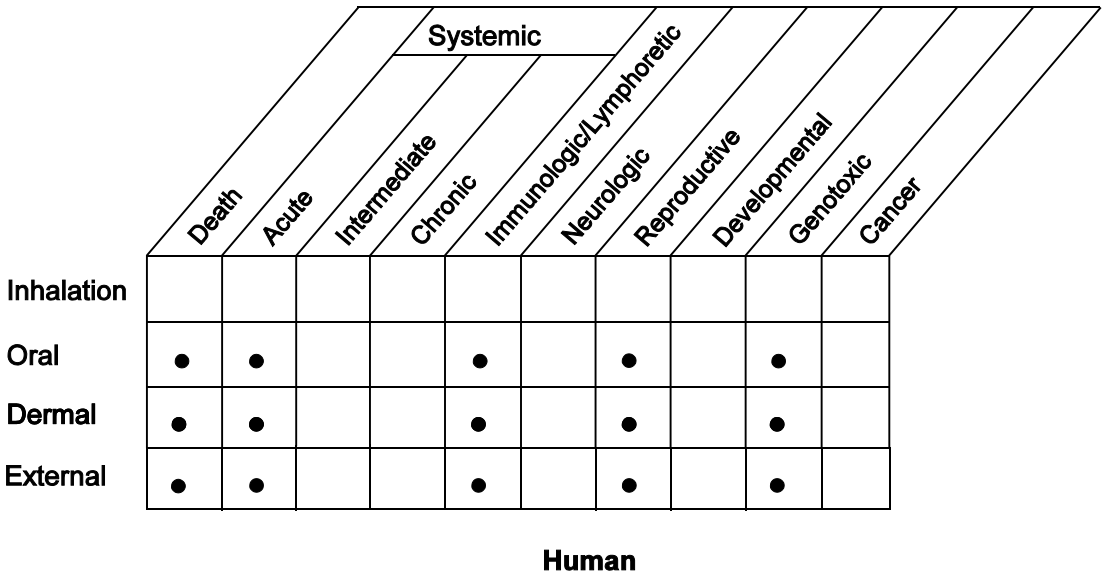
Human



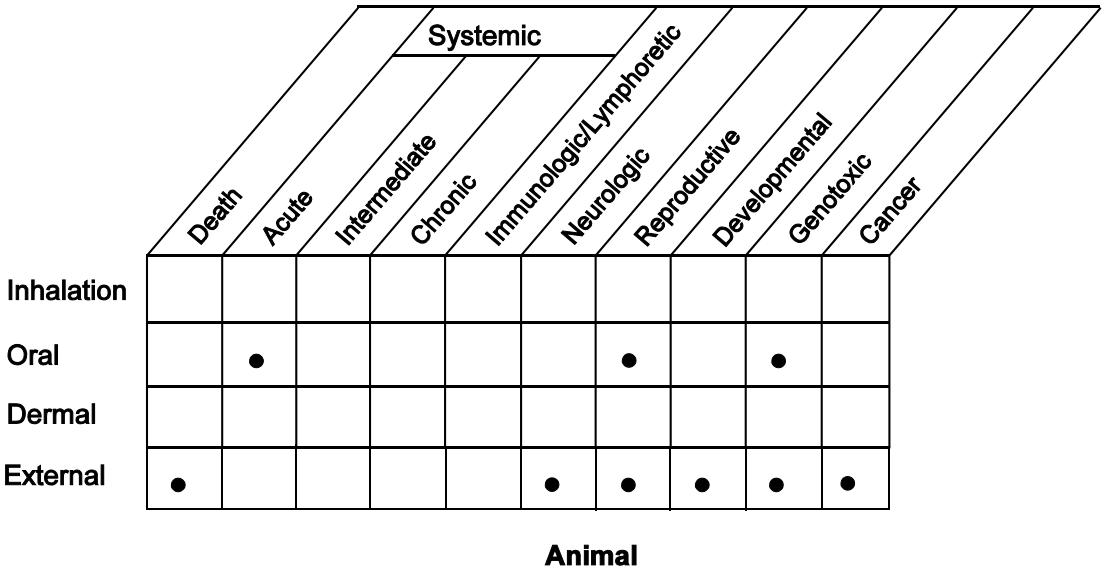
Animal

● Existing Studies

Figure 3-10. Existing Information on Health Effects of Radioactive Cesium



Human



Animal

● Existing Studies

3.12.2 Identification of Data Needs

Acute-Duration Exposure.

Stable Cesium. Results from human and animal studies indicate that stable cesium is of little acute oral toxicity concern. Toxicokinetic data regarding the widespread distribution of cesium absorbed following oral exposure indicate that dermal and inhalation exposure to stable cesium would not present a greater health concern than that posed by oral exposure. Acute-duration inhalation and oral MRLs were not derived for stable cesium due to a lack of human or animal data. To generate appropriate data for deriving acute-duration inhalation and oral MRLs for stable cesium, at least one comprehensive acute inhalation and one acute oral toxicity study would be needed of at least one animal species exposed to several dose levels.

Radioactive Cesium. Reports of adverse effects in humans that can be specifically attributed to acute exposure to radioactive cesium are restricted to the accounts of accidental exposure of a number of individuals to a ^{137}Cs source in which both external and internal (dermal and oral) exposure occurred (Brandão-Mello et al. 1991). Observed health effects were representative of those resulting from overexposure to any gamma emitting source of ionizing radiation. Limited animal data regarding acute oral exposure to radioactive cesium are limited to reports of dominant lethal mutations and reduced fertility in mice (Ramaiya et al. 1994). Acute-duration inhalation and oral MRLs were not derived for radioactive cesium due to a lack of human or animal data. To generate appropriate data for deriving acute-duration inhalation and oral MRLs for radioactive cesium, at least one comprehensive acute inhalation study and one acute oral toxicity study of at least one animal species exposed to several dose levels would be needed. Such studies could be designed to also generate data regarding potential age-related differences in toxicity. However, great danger would be posed to investigators considering the exposure of laboratory animals to radioactive cesium at levels great enough to cause significant adverse health effects. It has been shown that distribution patterns of ^{137}Cs are similar in animals exposed to relatively nontoxic levels of ^{137}CsCl by parenteral injection, inhalation exposure, or oral administration (Boecker et al.1969a; Stara 1965). Therefore, the results of the intravenous injection studies in dogs (Nikula et al. 1995, 1996) provide the most reasonable indication of health effects that would be expected in animals exposed by inhalation or oral administration. An acute-duration MRL that was derived by ATSDR (1999) for acute external exposure to ionizing radiation was considered to be appropriate as an acute-duration MRL for external exposure to ionizing radiation from a radioactive cesium source.

Intermediate-Duration Exposure.

Stable Cesium. A single study of intermediate-duration oral exposure in humans is available and indicates that the nervous system may be a target of toxicity for high-dose stable cesium (Neulieb 1984). Limited data indicate that intermediate-duration oral exposure of pregnant mice to stable cesium may adversely affect developing fetuses (Messiha 1988b, 1989b). Orally-administered stable cesium has also been shown to be genotoxic to female mice (Ghosh et al. 1990, 1991). Intermediate-duration inhalation and oral MRLs were not derived for stable cesium due to the paucity of human or animal data. To generate appropriate data for deriving intermediate-duration inhalation and oral MRLs for stable cesium, at least one comprehensive intermediate-duration inhalation and one intermediate-duration oral toxicity study of at least one animal species exposed to several dose levels would be needed. Such studies could be designed to also generate data regarding potential age-related differences in toxicity.

Radioactive Cesium. No human or animal data are available in which intermediate-duration inhalation or oral exposure to radioactive cesium can be associated with adverse human health effects. When humans intake high levels of radioactivity from a radiocesium source, as was the case of acute exposure in Goiânia, Brazil (Brandão-Mello et al. 1991), such exposures should be of value in assessing potential health hazards. Animal studies could be designed to assess the health effects associated with intermediate-duration exposure to radioactive cesium. Such studies could be designed to also generate data regarding potential age-related differences in toxicity.

Chronic-Duration Exposure and Cancer.

Stable Cesium. Since there are no studies pertaining to noncancer or cancer health effects in humans or animals following chronic-duration inhalation or oral exposure to stable cesium, no chronic-duration inhalation or oral MRLs were derived for stable cesium. Additional data from acute- and intermediate-duration animal studies might be helpful in determining the need for longer-term studies.

Radioactive Cesium. There are no data regarding noncancer or cancer health effects in humans following chronic-duration inhalation or dermal exposure to radioactive cesium. Low levels of radioactive cesium are found in the diets of individuals living in areas that have been contaminated with radioactive fallout; however, there is a lack of information regarding dose-response following chronic-duration oral exposure. No chronic-duration inhalation or oral MRLs were derived for radioactive cesium. A chronic-duration MRL that was derived by ATSDR (1999) for chronic external exposure to ionizing radiation was

appropriate as a chronic-duration MRL for external exposure to ionizing radiation from a radioactive cesium source. Long-term research into health effects associated with chronic exposure to radioactive cesium following incidents such as the Chernobyl nuclear accident may help to elucidate long-term non cancer and cancer health risks from chronic exposure to radionuclides of cesium.

Genotoxicity.

Stable Cesium. No genotoxicity studies of *in vivo* exposure of humans to stable cesium compounds were located. Stable cesium (as cesium chloride) induced chromosomal aberrations in human lymphocytes *in vitro* (Ghosh et al. 1993) and in mouse bone marrow *in vivo* (Ghosh et al. 1990, 1991). Cesium sulfate was not mutagenic in *E. coli* either with or without metabolic activation (Olivier and Marzin 1987). Studies in mammalian systems would be useful. Studies of workers exposed to known levels of stable cesium would be useful in establishing whether or not cesium is of genotoxicity concern in humans.

Radioactive Cesium. *In vivo* human data are limited to the findings of increased point mutations in T-lymphocytes and chromosomal aberrations among individuals who had been exposed to beta and gamma radiation from a ^{137}Cs source (Natarajan et al. 1998; Skandalis et al. 1997) and apparently increased frequencies of chromosomal aberrations in lymphocytes of children living in areas contaminated by ^{137}Cs fallout following the Chernobyl nuclear accident (Padovani et al. 1993). *In vitro* exposure of human lymphocytes to a ^{137}Cs source resulted in an increased frequency of micronuclei (Balasem and Ali 1991). *In vivo* oral and external exposure of male mice to ^{137}Cs resulted in increases in dominant lethal mutations (Ramaiya et al. 1994). Increases in the frequency of reciprocal translocations in spermatogonia were observed following oral exposure to ^{137}Cs in mice (Ramaiya et al. 1994) and external exposure to a ^{137}Cs source in crab-eating monkeys (Tobari et al. 1988). A number of *in vitro* genotoxicity assays have indicated chromosomal aberrations and breaks, sister chromatid exchanges, and micronuclei in animal cells (Arslan et al. 1986; Biedermann et al. 1991; Doggett and McKenzie 1983; Hintenlang 1993; Iijima and Morimoto 1991; Kamiguchi et al. 1991; Mikamo et al. 1990, 1991).

Human and animal studies show that external and internal exposure to radioactive cesium is a genotoxicity concern. External exposure to any gamma source would be expected to result in genotoxic effects similar to those observed following external exposure to radioisotopes of cesium (see ATSDR 1999 for more information on ionizing radiation). Whenever possible, additional human studies should focus on exposure levels to establish dose-response relationships.

Reproductive Toxicity.

Stable Cesium. There are no reports of reproductive effects in humans or animals exposed to stable cesium. Although stable cesium appears to be of relatively low toxicity concern, animal studies could be designed to assess the potential for adverse health effects (including reproductive effects) associated with repeated exposure. Human data could be collected from individuals occupationally-exposed to significant levels of stable cesium.

Radioactive Cesium. Reports of human reproductive effects following exposure to radioactive cesium are limited to the findings of reduced sperm counts following external and internal exposure to a ^{137}Cs source (Brandão-Mello et al. 1991). Mice, exposed to ^{137}Cs either orally or externally, exhibited reduced fertility (including complete sterility) (Ramaiya et al. 1994). Persistent germinal epithelium damage and azoospermia were observed in all long-term surviving dogs that had been administered intravenous injections of ^{137}Cs (Nikula et al. 1995, 1996). In cases of known human exposure to radioactive cesium, exposure-response relationships should be established when possible. Although reduced fertility has been shown in males, additional animal studies could be designed to assess the potential for reproductive toxicity in females.

Developmental Toxicity.

Stable Cesium. There are no reports of reproductive effects in humans exposed to stable cesium. One investigator reported reduced body weight and certain organ weights among offspring, as well as indications of altered activity of some hepatic enzymes among offspring of pregnant mouse dams repeatedly exposed (orally) to stable cesium (Messiha 1988b). The same investigator reported similar results in pups exposed (via their nursing mothers) only during lactation (Messiha 1989b). These studies did not include gross and histopathologic examination of the offspring. Well-designed animal studies could more completely assess potential for the developmental toxicity of stable cesium.

Radioactive Cesium. Although there are no reports of developmental effects in humans exposed specifically to radioisotopes of cesium, impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed to high levels of ionizing radiation during critical stages of neural development (Schull and Otake 1999; Schull et al. 1988). External exposure to sufficiently high doses of radiation from a radioactive cesium source would be expected to result in similar effects. *In utero* exposure of rat and mouse fetuses via whole body exposure of dams resulted in

developmental effects such as reduced postnatal body weight, impaired motor activity, morphological changes in the brain, increased aggressive behavior, reduced brain and head size, and retarded odontogenesis and palatal closure (Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994). Continued monitoring of populations known to have been exposed to ionizing radiation (including radioactive cesium sources) should help to refine estimates of dose-response and the relationship to adverse health effects, including developmental toxicity.

Immunotoxicity.

Stable Cesium. There are no reports regarding the immunotoxicity of stable cesium in humans or animals. Animal studies could be designed to assess these parameters, but such studies do not presently seem necessary.

Radioactive Cesium. Severe bone marrow depression was observed in individuals exposed externally and internally to a ^{137}Cs source. This effect is typical of individuals exposed to ionizing radiation (see ATSDR 1999 for additional information on the effects of ionizing radiation). A similar effect was observed in dogs given an intravenous injection of ^{137}Cs (Nikula et al. 1995). Data should be collected from individuals known to have been exposed to ionizing radiation (including that from radioactive cesium sources). Additional animal studies could be designed to establish dose-response relationships.

Neurotoxicity.

Stable Cesium. Data regarding neurological effects of stable cesium in humans are restricted to a single case of an investigator reporting feelings of euphoria, heightened sense perception, and tingling sensations within 15 minutes of ingesting oral doses of cesium chloride during a 36-day exposure period. No apparent adverse effects on mental or motor skills were observed (Neulieb 1984). Administration of cesium chloride to animals has triggered stimulant (Johnson 1972; Messiha 1978) and depressant (Bose and Pinsky 1981, 1983b, 1984; Bose et al. 1981; Pinsky et al. 1980) central nervous system responses. Additional animal studies could be designed to elucidate mechanisms responsible for the observed neurological effects.

Radioactive Cesium. Although there are no reports of neurotoxicity in humans exposed specifically to radioisotopes of cesium, impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed to high levels of external ionizing radiation during critical

stages of neural development (Schull and Otake 1999; Schull et al. 1988). External exposure to sufficiently high doses of radiation from a radioactive cesium source would be expected to result in similar effects. *In utero* exposure of rat and mouse fetuses via whole-body exposure of dams resulted in impaired motor activity, morphological changes in the brain, increased aggressive behavior, and reduced brain and head size (Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994), effects that have been shown to be related to critical developmental stages. Neurotoxic effects, noted in humans suffering from acute radiation syndrome due to ionizing radiation exposure, are well-characterized (see ATSDR 1999 for more detailed information on the effects of ionizing radiation). Such effects would be expected in humans exposed to sufficiently high doses of radiation from a radioactive cesium source. Additional well-designed animal studies might elucidate mechanisms of neurotoxicity, but do not presently appear to be needed.

Epidemiological and Human Dosimetry Studies.

Stable Cesium. Stable cesium is ubiquitous in the earth's crust, but is found at such low environmental levels that the probability of human intake of toxic amounts of stable cesium is negligible. Although there is no apparent need for specifically designed epidemiological or human dosimetry studies regarding stable cesium, such data, collected from individuals occupationally exposed to significant amounts of cesium or persons living near toxic waste sites with significant levels of cesium, might be useful.

Radioactive Cesium. Due to accidental or intentional releases during nuclear fission, ^{134}Cs and ^{137}Cs can be found in air, soil, water, and food. There is concern for the health of humans living in close proximity to release or storage sites or in areas receiving significant amounts of radioactive fallout. Epidemiological studies of radiation dose typically involve estimates of exposure that are based on whole-body measurements of internally-deposited ^{134}Cs or ^{137}Cs or genotoxic effects such as chromosomal aberrations in peripheral blood lymphocytes. A need remains for epidemiological data that can provide quantitative human dose-response information while supplying additional information on the health effects of exposure to ionizing radiation and radioisotopes of cesium; in particular, for cases of known internal exposure.

Biomarkers of Exposure and Effect.

Exposure. Both stable and radioactive isotopes of cesium may be detected in samples of urine, blood, feces, or body tissues. Due to the relatively long biological half-time of cesium (several months in humans), short-term exposures cannot be readily distinguished from longer-term ones. No new biomarkers of exposure are needed at this time.

Effect. No known biomarkers of effect exist for stable cesium. Although high radiation doses from internally deposited radioactive cesium can cause bone marrow aplasia, altered blood values, and increased chromosomal aberrations in lymphocytes (Brandão-Mello et al. 1991; Natarajan et al. 1998), these effects are not specific to radioactive cesium.

Absorption, Distribution, Metabolism, and Excretion. Human and animal data show that inhaled or ingested cesium (in soluble compounds) is rapidly absorbed into the blood (Boecker 1969a, 1969b; Henrichs et al. 1989; Lie 1964; Miller 1964; Stara 1965; Stara and Thomas 1963), whereas relatively insoluble forms of cesium are not readily absorbed following inhalation or oral exposure (Boecker et al. 1974, 1977; Leroy et al. 1966; Talbot et al. 1993). Dermal absorption has been qualitatively (but not quantitatively) demonstrated in rats (Pendic and Milivojevic 1966). Additional studies could measure relative absorption rates for a variety of soluble and insoluble cesium compounds. Furthermore, studies could be designed to measure dermal absorption rates. Other studies could further assess the fate of relatively insoluble inhaled particles containing radioisotopes of cesium that may be retained in lung tissue for long periods of time.

Cesium absorbed via inhalation or ingestion has been shown to be rapidly distributed throughout the body of humans and animals (Boecker 1969a, 1969b; Furchner et al. 1964; Lie 1964; Miller 1964; Rosoff et al. 1963; Stara 1965; Stara and Thomas 1963). Once absorbed by pregnant women, cesium can pass the placental barrier and be absorbed by the conceptus. Absorbed cesium can also be found in the milk of lactating women (Mahlum and Sikov 1969; Vandecasteele et al. 1989). Once cesium is absorbed into body fluids, distribution patterns in soft tissue are expected to be similar for any route of exposure since cesium is distributed throughout the body as the cation (Cs^+), much like potassium (K^+). Available studies appear to adequately describe the distribution of absorbed cesium. Additional studies could be designed to elucidate mechanisms whereby cesium ions may influence central nervous system activity.

Human and animal studies adequately describe elimination of absorbed cesium, primarily via the urine (Boecker 1969b; Iinuma et al. 1965; Rosoff et al. 1963; Stara 1965; Stara and Thomas 1963). Age-related differences in elimination rates have been described in humans (Boni 1969b; Melo et al. 1994) and dogs (Melo et al. 1996). In cases of known human exposure to cesium, additional information may help to further assess age-related differences in the toxicokinetics of cesium.

Comparative Toxicokinetics. Available cesium toxicokinetic data in humans and various animal species indicate similar patterns of absorption, distribution, and elimination. The central nervous system appears to be a target for effects in humans (Neulieb 1984) and animals (Bose and Pinsky 1981, 1983b, 1984; Bose et al. 1981; Johnson 1972; Messiha 1978; Pinsky et al. 1980). Additional studies could be designed to elucidate and compare mechanisms responsible for central nervous system effects.

Methods for Reducing Toxic Effects. Oral administration of Prussian blue that exchanges potassium for cesium, cathartics that shorten the transit time of ingested cesium within the gastrointestinal tract, and gastric lavage may aid in reducing peak absorption of ingested cesium, but due to the rapid absorption of cesium from soluble cesium compounds, these measures would only be of potential benefit within a short time following initial exposure. Most countermeasures focus on reducing the body burden of absorbed cesium. The intestinal reabsorption of cesium that is excreted into the small intestine via the bile can be blocked by oral administration of Prussian blue, forming an insoluble cesium complex that is excreted in the feces. Animal studies also indicate that increased plasma concentrations of potassium may increase the mobilization of cesium from tissues, increasing its excretion (Richmond and Furchner 1961). There are no prescribed methods for interfering with mechanisms of action for toxic effects of cesium, since such mechanisms have not been elucidated.

Children's Susceptibility. Available information on age-related differences in health effects comes from *in utero* exposure to radiation from external ionizing radiation sources (including radioisotopes of cesium). Studies have shown neurological effects in humans and animals exposed during critical periods of central nervous system development (Koshimoto et al. 1994; Minamisawa et al. 1990; Norton and Kimler 1987, 1988; Schull and Otake 1999). Comparative studies of neurological effects in animals first irradiated as juveniles or adults are lacking. No information was located regarding age-related health effects in humans or animals exposed to stable cesium.

PBPK models account for potential age-related differences in deposition of inhaled cesium, as well as differences in elimination rates for absorbed cesium (see Section 3.4.5 for more information on PBPK

models). Potential differences in absorption rates of inhaled cesium compounds could be due to age-related differences in physical properties of the respiratory system and/or ventilation patterns. PBPK models also could adjust for potential age-related differences in gastrointestinal absorption. If such differences exist, they would likely be the result of differences in diffusion rates and active transport mechanisms. Young children exhibit biological half-times for absorbed cesium that are shorter than those of older children and adults (Boni 1969b; Melo et al. 1994); these age-related differences are related to body mass and could be the result of age-related differences in tissue retention and excretory rates.

Cesium is found in the breast milk of mothers with an internal cesium burden, and can be transferred to nursing infants (Johansson et al. 1998). Cesium has been shown to cross the placental barrier of animals, but concentrations of cesium in the fetal tissues are less than those in corresponding tissues of the mother (Mahlum and Sikov 1969; Vandecasteele et al. 1989). Data collected from areas containing elevated concentrations of radioactive cesium fallout might be of value in further assessing age-related transfer rates.

Biomarkers of exposure or effect are the same in adults and children (see Section 3.8 for detailed information on biomarkers of exposure and effect). There are no data on interactions of cesium with other chemicals in children. No pediatric-specific methods have been found to reduce peak absorption, or body burden, of cesium following exposure, although methods employed to reduce the body burden of cesium in adults are also effective in children.

Child health data needs relating to exposure are discussed in 6.8.1 Identification of Data Needs: Exposures of Children.

3.12.3 Ongoing Studies

A limited number of ongoing studies, mainly concerned with the electrolytic basis of muscle cell activity or therapeutic uses of radioactive cesium, were identified in the Federal Research In Progress database (FEDRIP 2000). These studies are summarized below.

Dr. D. Kass, from Johns Hopkins University, Baltimore, Maryland, is using cesium chloride provocation studies to measure repolarization abnormalities in cardiac tissues of dogs experiencing ventricular arrhythmia.

r. E. Van Lunteren, from the Department of Veterans Affairs Medical Center, Cleveland, Ohio, is using cesium as a potassium channel blocker in frog skeletal muscle to test the hypothesis that potassium channels regulate contractile force and fatigability of skeletal muscle by altering resting membrane potential and/or action potential duration.

Dr. K. Suthanthiran, from Best Industries, Inc., Springfield, Ohio, is studying the feasibility of commercially manufacturing ^{131}Cs seeds and films (half-life 9.7 days) for use in cancer therapy.

4. CHEMICAL AND PHYSICAL INFORMATION

4.1 CHEMICAL IDENTITY

Information regarding the chemical identity of cesium is located in Table 4-1.

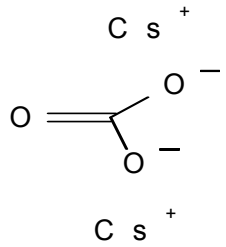
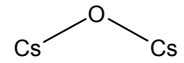
4.2 PHYSICAL AND CHEMICAL PROPERTIES

Information regarding the physical and chemical properties of cesium and selected cesium compounds are located in Table 4-2.

Cesium is a silvery white, soft, ductile metal with only one (+1) oxidation state. At slightly above room temperature, cesium exists in the liquid state. Compared to the other stable alkali metals, cesium has the lowest boiling point and melting point, the highest vapor pressure, the highest density, and the lowest ionization potential. These properties make cesium far more reactive than the other members of the alkali metal group. When exposed to air, cesium metal ignites, producing a reddish violet flame and forms a mixture of cesium oxides. Pure cesium reacts violently with water to form cesium hydroxide, the strongest base known, as well as hydrogen gas. The burning cesium can ignite the liberated hydrogen gas and produce an explosion. Cesium salts and most cesium compounds are generally very water soluble, with the exception of cesium alkyl and aryl compounds, which have low water solubility.

There are several radioactive isotopes of cesium ranging from ^{114}Cs to ^{145}Cs (Helmers 1996). The radioactive isotopes have a wide range of half-lives ranging from about 0.57 seconds (^{114}Cs) to about $3x10^6$ years (^{135}Cs) (Helmers 1996). The radioactive isotopes ^{137}Cs and ^{134}Cs are significant fission products because of their high fission yield, their long half-lives, and their biochemical similarity to potassium. The fission yield of ^{137}Cs in nuclear reactions is relatively high, about 6 atoms of ^{137}Cs are produced per 100 fission events (WHO 1983). ^{137}Cs has a radioactive half-life of about 30 years and decays by beta decay either to stable ^{137}Ba or a meta-stable form of barium (^{137m}Ba). The meta-stable isotope (^{137m}Ba) is rapidly converted to stable ^{137}Ba (half-life of about 2 minutes) accompanied by gamma ray emission whose energy is 0.662 MeV (ICRP 1983). Figure 4-1 illustrates this decay scheme. The first beta decay mode that forms ^{137m}Ba accounts for roughly 95% of the total intensity, while the second mode accounts for about 5% (WHO 1983). Radioactive ^{134}Cs primarily decays to stable ^{134}Ba by beta decay accompanied by gamma ray emissions or less frequently to stable ^{134}Xe by electron capture (EC)

Table 4-1. Chemical Identity of Cesium and Compounds

Characteristic	Cesium (metal)	Cesium chloride	Cesium carbonate	Cesium hydroxide	Cesium oxide
Synonym(s)	Caesium	Cesium monochloride	Dicesium salt	Cesium hydrate	
Registered trade name(s)	No data	No data	No data	No data	No data
Chemical formula	Cs	CsCl	Cs_2CO_3	CsOH	Cs_2O
Chemical structure	Cs	Cs—Cl		Cs—OH	
Identification numbers:					
CAS registry	7440-46-2	7647-17-8	534-17-8	21351-79-1	20281-00-9
NIOSH RTECS	FK9225000	FK9625000	FK9400000	FK9800000	No data
EPA hazardous waste	No data	No data	No data	No data	No data
OHM/TADS	No data	No data	No data	No data	No data
DOT/UN/NA/IMCO shipping	No data	No data	No data	No data	No data
HSDB	No data	No data	No data	No data	No data
NCI	No data	No data	No data	No data	No data

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; RTECS = Registry of Toxic Effects of Chemical Substances

Table 4-2. Physical and Chemical Properties of Cesium and Compounds[a]

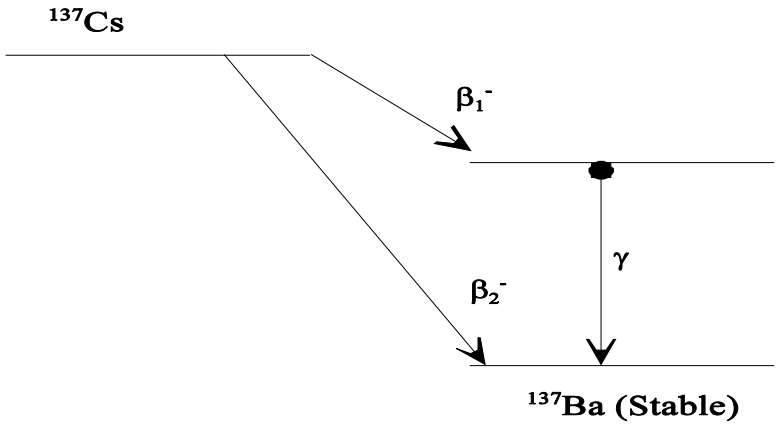
Property	Cesium (metal)	Cesium chloride	Cesium carbonate	Cesium hydroxide	Cesium oxide
Molecular weight	132.906	168.36	325.82	149.91	281.81
Color	Silvery-white	White	White	Colorless	Golden-yellow
Physical state	Solid (Liquid slightly above room temperature)	Solid	Solid	Solid	Solid
Melting point	29 EC	646 EC	610 EC	272 EC	490 EC
Boiling point	685 EC	1290 EC	No data	No data	No data
Density, g/cm^3	1.93 (20 EC)[b]	3.988 (20 EC)[b]	4.24 (20 EC)[b]	3.68 (20 EC)[b]	4.65 (20EC)[b]
Odor	No data	No data	No data	No data	No data
Odor threshold:					
Water	No data	No data	No data	No data	No data
Air	No data	No data	No data	No data	No data
Solubility:					
Water	Reacts violently with water	1.87 kg/L (20 EC)	2.1 kg/L	4 kg/L (15 EC)	Very soluble in water[b]
Organic-solvent(s)		Soluble in ethanol[b]	Soluble in ethanol and ether[b]	Soluble in ethanol[b]	
Partition coefficients:					
Log K_{ow}	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable
Log K_{oc}	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable
Vapor pressure	0.0075 mmHg at 144.5 EC	No data	No data	No data	No data
Henry's law constant	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable

Table 4-2. Physical and Chemical Properties of Cesium and Compounds[a] (*continued*)

Property	Cesium (metal)	Cesium chloride	Cesium carbonate	Cesium hydroxide	Cesium oxide
Autoignition temperature	No data	No data	No data	No data	No data
Flashpoint	No data	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data	No data
Conversion factors	No data	No data	No data	No data	No data
Explosive limits	No data	No data	No data	No data	No data

[a]Data from Burt 1993 unless otherwise specified.
[b]Lide 1998

Figure 4-1. The Decay Scheme of ^{137}Cs



β = beta decay; γ = γ-ray emission

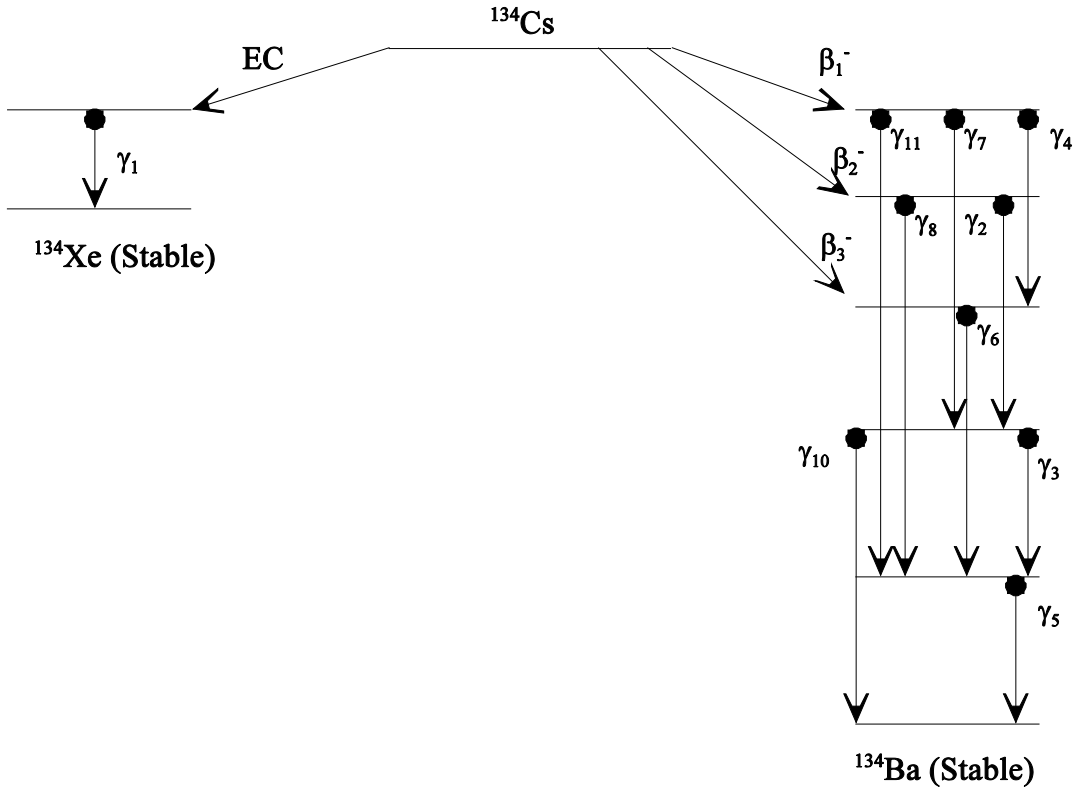
accompanied by a single gamma ray emission as depicted in Figure 4-2. The energy of the various gamma rays are in the range of 0.24–1.4 MeV. The half-life average energy of the beta transitions and intensity of the transitions for both ^{134}Cs and ^{137}Cs are summarized in Table 4-3.

Table 4-3. Decay Properties of the Radioactive Isotopes of Cesium[a]

Isotope	Half-life (years)	Decay mode	Intensity percent	Beta particle energy (Mev)
^{134}Cs	2.062	β_1^-	27	0.02309
		β_2^-	2.5	0.1234
		β_3^-	70	0.2101
^{137}Cs	30	β_1^-	94.6	0.1734
		β_2^-	5.4	0.4246

[a]ICRP 1983
[b]The ^{134}Xe daughter yield from the electron capture decay of ^{134}Cs is approximately $3x10^{-6}$.

Figure 4-2. The Decay Scheme of ^{134}Cs



EC = Electron Capture; β = beta decay; γ = γ-ray emission

5. PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

5.1 PRODUCTION

Cesium is the rarest of the naturally occurring alkali metals, ranking 40th in elemental abundance. Although it is widely distributed in the earth's crust, cesium is found at relatively low concentrations. Granites contain cesium at about 1 ppm and sedimentary rocks contain approximately 4 ppm. Cesium is found in muscovite, beryl, spodumene, potassium feldspars, leucite, petalite, and other related minerals. The most common commercial source of cesium is pollucite, which contains between 5 and 32% Cs_2O (Burt 1993). There are three basic methods of converting pollucite ore to cesium metal or related compounds: direct reduction with metals; decomposition with bases; and acid digestion. In each method, grinding of the mined ore to approximately 75 μm sized particles precedes chemical conversion.

Direct Reduction With Metals. Pollucite is directly reduced by heating the ore in the presence of calcium to 950 EC in vacuum, or in the presence of either sodium or potassium to 750 EC in an inert atmosphere (Burt 1993). Excessive amounts of reducing metal are required and the resultant cesium metal is impure, requiring further distillation and purification.

Decomposition With Bases. Alkaline decomposition is carried out by roasting the pollucite ore with either a calcium carbonate-calcium chloride mix at 800–900 EC or with a sodium carbonate-sodium chloride mix at 600–800 EC followed by a water leach of the roasted mass. The resultant cesium chloride solution is separated from the gangue by filtration (Burt 1993).

Acid Digestion. Acid digestion of pollucite is the preferred commercial process for producing pure cesium. Hydrofluoric, hydrobromic, hydrochloric, and sulfuric acid may be employed in this method. Hydrofluoric and hydrobromic acid digestion yield the greatest cesium recovery, but the inherent difficulties of safely handling these acids limit their use. Digestion with hydrochloric acid takes place at elevated temperatures and produces a solution of mixed cesium chlorides, aluminum, and other alkali metals separated from the siliceous residue by filtration. The impure cesium chloride can be purified as cesium chloride double salts that are then recrystallized. The purified double salts are decomposed to cesium chloride by hydrolysis or precipitated with hydrogen sulfide (Burt 1993).

Sulfuric acid digestion is performed at 110 EC with a 35–40% solution of sulfuric acid, followed by a hot water wash and vacuum filtration. Cesium alum is crystallized from the leach filtrate by stage cooling to

50 EC and then 20 EC and roasted in the presence of 4% carbon. The residue is leached to produce cesium sulfate solution, which can be converted to cesium chloride (Burt 1993).

Radioactive isotopes of cesium such as ^{134}Cs and ^{137}Cs are produced by nuclear fission in fuel rods in nuclear power plants and in fallout from nuclear weapons. Radiocesium can be recovered from fission products by digestion with nitric acid. After filtration to remove the waste, the radioactive cesium phosphotungstate is precipitated using phosphotungstic acid (Burt 1993). Other processes for the removal of ^{134}Cs and ^{137}Cs from radioactive waste involve solvent extraction using macrocyclic polyethers, or crown ethers and coprecipitation with sodium tetraphenylboron (Burt 1993).

5.2 IMPORT/EXPORT

The United States is 100% import-reliant for cesium. There are no salient statistics such as production volume, consumption, or import/export volumes of cesium. Although there is no information regarding the countries shipping cesium or cesium compounds to the United States, it is believed that Canada is the major source of cesium (USGS 1999). Other possible sources of cesium-bearing material include Germany and the United Kingdom.

5.3 USE

There are relatively few commercial uses for cesium metal and its compounds. Cesium is used as a getter (a getter combines chemically with residual gas in partial vacuum in order to increase the vacuum) for residual gas impurities in vacuum tubes and as a coating to reduce the work function of the tungsten filaments or cathodes of the tubes. Cesium iodide and cesium fluoride are used in scintillation counters, which convert energy from ionizing radiation into pulses of visible light (Burt 1993). Cesium is also used in magnetohydrodynamic power generators as a plasma seeding agent (Lewis 1997). Recently, cesium compounds have been employed as catalysts in organic synthesis, replacing sodium or potassium salts. One of the most interesting uses of cesium is in the production of highly accurate atomic clocks. When exposed to microwave radiation, the natural vibration of cesium atoms occurs at a frequency of 9,192,631,770 Hz, and 1 second in time is defined as the duration of 9,192,631,770 periods of radiation absorbed or emitted by the transition of ^{133}Cs atoms in two hyperfine levels of their ground state. Radioactive ^{137}Cs has been approved as the gamma ray source for the sterilization of wheat, flour, potatoes, surgical equipment and other medical supplies, and sewage sludge, and is also used as a calibration source in gamma ray spectroscopy (Lewis 1997).

5.4 DISPOSAL

Because of its high reactivity, special precautions are required for the handling and disposal of pure cesium metal. Cesium metal is usually stored and transported in stainless steel containers, which are contained in outer packing, to ensure that the metal remains in a dry, oxygen-free environment.

Most nonradioactive cesium minerals, compounds, and materials do not require special disposal and handling requirements. However, some chemical forms may be classified as hazardous materials if the compound is chemically reactive, flammable, or toxic. Care should be taken to read and understand all of the hazards, precautions, and safety procedures for each specific chemical form. In addition, all federal, state, and local laws and regulations should be investigated and subsequently followed with regard to disposal and handling of the specific chemical form of the cesium mineral, compound, or material.

Radioactive cesium requires special disposal and handling requirements. Radioactive waste containing ^{134}Cs and ^{137}Cs is usually grouped into four categories: low-level waste (LLW), high-level waste (HLW), mixed waste, and spent nuclear fuel.

Low-level waste is all radioactive waste that cannot be classified as either HLW, spent fuel, or mixed waste. Low-level does not necessarily mean low radioactivity or low environmental hazard. Low-level waste types that may be contaminated with ^{134}Cs and ^{137}Cs include both wet and dry wastes. Examples of the physical form of LLW are: spent ion exchange resins, filter sludges, filter cartridges, evaporator bottoms, compactible trash, non-compactible trash, irradiated components, ashes produced from the incineration of combustible material, contaminated detergents or solvents, organic liquids, and discarded contaminated equipment or tools. Of the LLW generated today, approximately 64% of the volume and 70% of the radioactivity is generated as a result of nuclear power plant activities or supporting fuel cycle operations. Other sources of LLW are commercial, academic, and government research laboratories and medical facilities. Radiocesium contamination accounts for only a small fraction of the activity of LLW. Nearly all of the ^{134}Cs and ^{137}Cs produced as a result of fission events remains trapped within the spent nuclear fuel rods (DOE 1996b). Low-level waste from Department of Energy (DOE) sources is currently disposed of at several DOE facilities across the United States. Only two sites accept non-DOE LLW: Barnwell, South Carolina and Richland, Washington (DOE 1996a). As required by the Federal Low Level Radioactive Waste (LLRW) Policy Act in 1980 and the 1985 amendments, states are required to build facilities to contain LLW generated from sources within its boundaries. The law encourages states to cooperate together in coordinating LLW disposal facilities. Many states have formed “compacts” to

collaborate on construction of these LLW facilities. However, other than Barnwell, South Carolina and Richland, Washington, no other facility in the United States is accepting LLW from non-DOE sources (Eisenbud 1987). Over half of the LLW in the eastern United States is disposed of at the Barnwell site (Eisenbud 1987). The method of disposal for LLW has been to package the material in drums or boxes and bury the material in shallow pits and trenches. Approximately 3 million cubic meters of LLW generated has been disposed of IN this way (DOE 1996a).

As defined by the Nuclear Waste Policy Act, HLW is "the highly radioactive material resulting from the reprocessing of spent nuclear fuel, including liquid waste produced directly in reprocessing and any solid material derived from such liquid waste that contains fission products in sufficient concentration..." (42 USC 100). Most HLW has been generated from the production of plutonium. A smaller fraction is related to the recovery of enriched uranium from naval reactor fuel. This waste typically contains highly concentrated solutions of relatively long-lived fission by-products such as ^{90}Sr and ^{137}Cs, hazardous chemicals, and toxic heavy metals. Liquid HLW is typically stored in large underground tanks of either stainless steel or carbon steel depending on whether they are acid or alkaline solutions. Approximately 100 million gallons of liquid HLW is stored in underground tanks in Washington, South Carolina, Idaho, and New York. These tanks contain a variety of radioactive liquids, solids, and sludges. Some of the liquid waste has been solidified into glass, ceramic slag, salt cake, and sludge. High-level waste in solid form is stored in underground bins (DOE 1996a).

Mixed waste contains both radioactive and chemically hazardous materials. All HLW is managed as mixed waste and some LLW is classified as mixed waste. Certain hazardous mixed wastes that contain radioactive isotopes have been incinerated in Oak Ridge, Tennessee (DOE 1996a). Spent nuclear fuel, such as fuel elements and irradiated targets used in a nuclear reactors, are currently stored at the commercial nuclear power plants and DOE facilities where they were produced. Spent fuel is highly radioactive, due to the large concentration of fission products, and must be stored in special water-cooled pools that shield and cool the material. Nearly all of the DOE spent fuel, about 3,000 metric tons, is stored at four sites: Hanford, Savannah River, Idaho National Environmental and Engineering Laboratory, and West Valley. Commercial reactors have generated more than 30,000 metric tons of spent fuel. The spent fuel from these facilities is located at >100 commercial nuclear reactor sites around the country.

The establishment of a HLW and spent fuel repository for both DOE and commercial waste is currently under evaluation at Yucca Flats, Nevada. This commercial waste storage facility will not be ready to accept spent fuel before 2010.

6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

Stable cesium has been identified in at least 10 of the 1,585 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2001). It was reported that ^{134}Cs has been identified in at least 3 of the 1,585 hazardous waste sites and ^{137}Cs has been identified in at least 22 of the 1,585 hazardous waste sites proposed for inclusion on the EPA NPL. However, the number of sites evaluated for cesium is not known. The frequency of these sites within the United States can be seen in Figures 6-1, 6-2, and 6-3. Of these sites, none are located in the Commonwealth of Puerto Rico.

Naturally-occurring cesium and cesium minerals consist of only one stable isotope, ^{133}Cs. Cesium occurs in the earth's crust at low concentrations. Granites contain an average cesium concentration of about 1 ppm and sedimentary rocks contain about 4 ppm (Burt 1993). Higher concentrations are found in lepidolite, carnallite, muscovite, beryl, spodumene, potassium feldspars, leucite, petalite, and related minerals. The most important source of commercial cesium is the mineral pollucite, which usually contains about 5–32% Cs_2O (Burt 1993). The largest deposits of pollucite are located in Manitoba, Canada and account for about two-thirds of the world's known supply. Smaller deposits are located in Zimbabwe, Namibia, Brazil, Scandinavia, Czechoslovakia, and the United States. Continental dust and soil erosion are the main emission sources of naturally occurring cesium present in the environment. Cesium is also released to the environment as a result of human activities. The mining of pollucite ores and the production and use of cesium compounds in the electronic and energy production industries contribute to its direct release to the environment. Cesium has also been detected in the fly ash of hazardous waste incinerators and coal burning power plants (Fernandez et al. 1992; Mumma et al. 1990). Since the production and use of cesium compounds are limited, and since the natural concentration of cesium in the earth's crust is low, ^{133}Cs is not often monitored or detected in the environment.

Of much greater concern is the release of radioactive forms of cesium to the environment, such as ^{137}Cs and ^{134}Cs. These and other radioactive isotopes were released to the environment as a result of atmospheric testing of nuclear weapons (carried out from 1945 to 1980) and accidents that occurred at nuclear power plants such as the incident at the Chernobyl nuclear power plant in 1986 and the accident at the Windscale nuclear weapons facility in the United Kingdom in 1957. Small amounts of ^{137}Cs and ^{134}Cs are also released in the airborne and liquid effluents during the normal operation of nuclear power

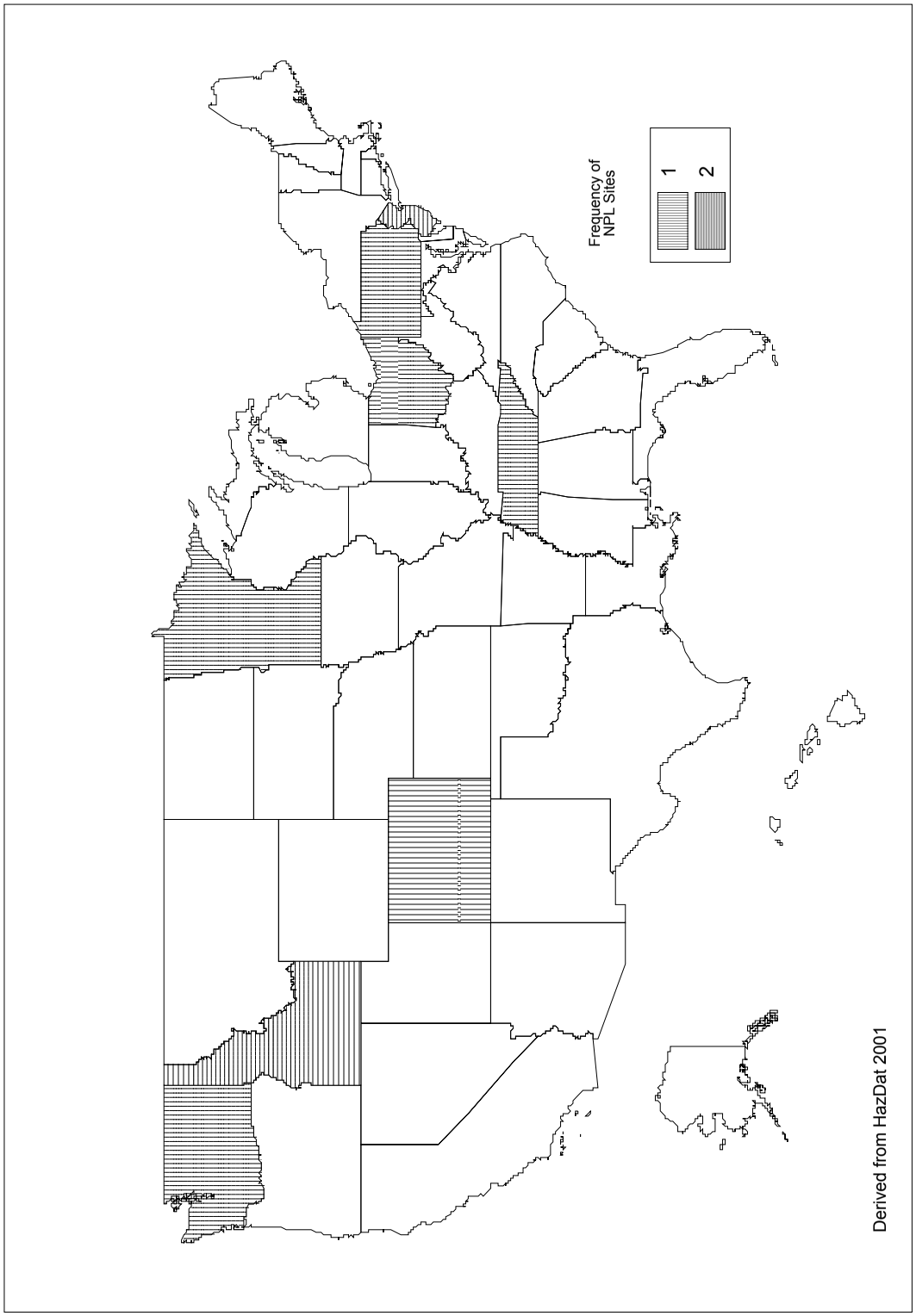


Figure 6-1. Frequency of NPL Sites with Cesium Contamination

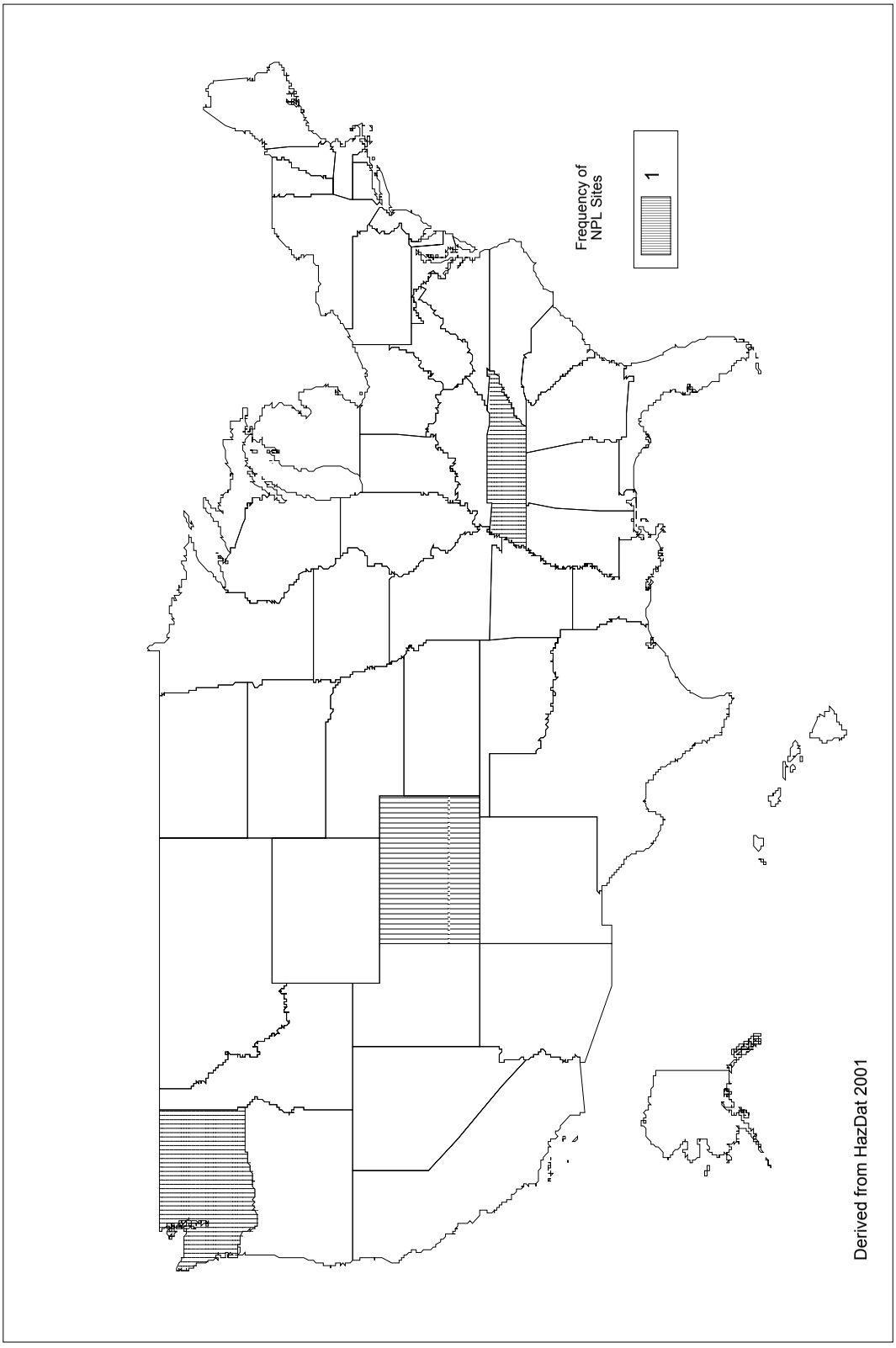


Figure 6-2. Frequency of NPL Sites with Cesium 134 Contamination

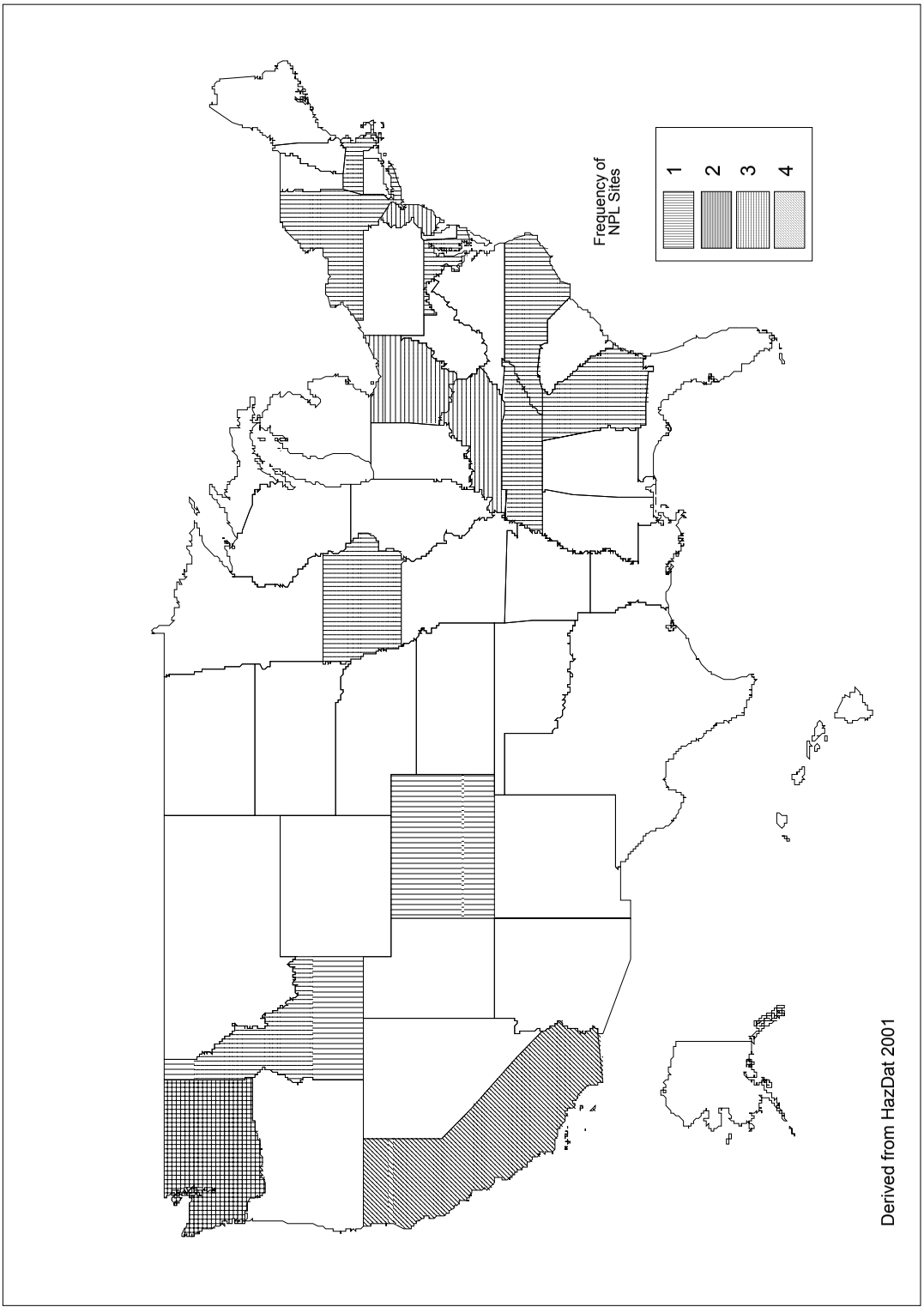


Figure 6-3. Frequency of NPL Sites with Cesium 137 Contamination

plants. These levels are very low in comparison to the amounts released from weapons tests and accidents at nuclear power plants and are not expected to have a major impact upon human health. For the most part, testing of nuclear weapons has been discontinued by most nations for many years now. However, India and Pakistan have recently (May of 1998) conducted limited underground tests of nuclear weapons (UN 1998).

Radioactive material is commonly referred to by its activity rather than its mass. The activity is the number of disintegrations that the material undergoes in a given period of time. The most common units of activity are the curie (Ci) or the becquerel (Bq). One curie is equal to $3.7x10^{10}$ disintegrations per second (dps) and 1 becquerel is equal to 1 dps. For convenience, picocuries (pCi) are often reported for lower activities; $1pCi=1x10^{-12}$ Ci (see Section 1.2).

Radioactive cesium is removed from the air by wet and dry deposition and can travel thousands of miles before settling to earth. Wet deposition is considered the most important pathway for the removal of radioactive cesium from the atmosphere. It is a complex process that depends upon meteorological conditions such as temperature, the microphysical structure of the clouds, and the rainfall rate, as well as the physical and chemical properties of the airborne cesium.

Cesium has very low mobility in soil. In general, it has been reported that cesium usually does not migrate below depths of about 40 cm, with the majority of cesium being retained in the upper 20 cm of the soil surface (Korobova et al. 1998; Takenaka et al. 1998). Clay minerals and soils rich in exchangeable potassium adsorb cesium by binding the cations to interlayer positions of the clay particles (Paasikallio 1999). The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays. These factors can limit the uptake of cesium in grass and plant material. There are exceptional areas, however, where cesium fixation in soil is much less, resulting in greater transport in the soil and uptake in plants. Regions in Venezuela, Brazil, and Russia have been identified where the mobility of cesium is considerably greater than in other soils (LaBrecque and Rosales 1996; WHO 1983). Cesium is also deposited on plants and trees by wet and dry deposition and is absorbed into the flora through its foliage (Sawidis et al. 1990). The deposited cesium can make its way to soil through decomposition of the contaminated foliage.

Since the half-life for some radioactive isotopes of cesium is long (the half-life of ^{137}Cs is about 30 years and the half-life of ^{134}Cs is about 2 years), the general population is exposed to ^{137}Cs and ^{134}Cs for long periods of time after it is released from a nuclear accident or weapons test, with the greatest exposure

occurring near the source. Although inhalation and dermal exposure is possible, oral ingestion of contaminated food items is the greatest source of internal exposure for both naturally occurring and radioactive cesium. Cesium is uniformly distributed throughout the whole body similar to potassium, and it does not accumulate in any one particular part of the body like iodine (thyroid) or strontium (bones). For this reason, radioactive cesium only poses significant health risks if a large amount has been ingested. Workers employed in the mining and milling of pollucite ores and the production of cesium compounds are exposed to cesium through oral, dermal, and inhalation routes. Similar routes of exposure to ^{137}Cs and ^{134}Cs occurs for workers employed in the nuclear industry. External exposure to gamma radiation can also occur for workers employed in the nuclear industry as well as for the general population. The health consequences of external exposure to gamma radiation are not unique to ^{137}Cs and ^{134}Cs, but are similar for all gamma emitting radionuclides.

6.2 RELEASES TO THE ENVIRONMENT

Throughout this chapter, the units used to express concentration or intake of cesium are the same units reported by the authors. In most cases, values are expressed in mass units when referring to ^{133}Cs, while radioactive cesium isotopes are expressed in units of activity.

According to the Toxic Release Inventory (TRI), in 1999, there were no reportable releases of ^{133}Cs, or its compounds into the environment by commercial sources (TRI99 2001). The TRI data should be used with caution since only certain types of facilities are required to report. Therefore, it is not an exhaustive list. Facilities are required to report to TRI if they have 10 or more full-time employees, or if the facility is classified under Standard Industrial Classification (SIC) codes 20–39, if the facility manufactures or processes over 25,000 pounds of the chemical, or otherwise uses more than 10,000 pounds of the chemical in a calendar year.

In the United States, commercial nuclear power plant operators are required to report any detectable quantities of radioactive materials released to the environment (10 CFR 50.36a). Table 6-1 summarizes releases of ^{137}Cs and ^{134}Cs to the atmosphere and water for 1993 from pressurized water reactor (PWR) and boiling water reactor (BWR) nuclear power plants. Nearly all of the radioactive material reported as being released in effluents, are from planned releases. Planned releases result from normal plant operation or from anticipated operational occurrences. The latter include unplanned releases of radioactive materials from miscellaneous actions such as equipment failure, operator error, or procedure error; these releases are not of such consequence as to be considered an accident.

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Boiling water reactors					
Browns Ferry[b]	Decatur, AL	0.033	0.18	3.5×10^{-4}	8.9×10^{-4}
Brunswick[b]	Wilmington, NC	2.9×10^{-4}	1.7×10^{-3}	No data	7.0×10^{-4}
Clinton	Clinton, IL	No data	No data	No data	No data
Cooper	Omaha, NE	9.3×10^{-3}	0.052	No data	No data
Dresden[a]	Joliet, IL	1.2×10^{-6}	0.025	No data	1.9×10^{-4}
Duane Arnold	Cedar Rapids, IA	No data	No data	No data	1.4×10^{-6}
Edwin I. Hatch	Baxley, GA	6.3×10^{-3}	0.044	No data	6.2×10^{-5}
Fermi	Laguna Beach, MI	No data	8.3×10^{-6}	No data	No data
Grand Gulf	Vicksburg, MS	3.5×10^{-4}	6.0×10^{-4}	2.1×10^{-6}	2.3×10^{-6}
Hope Creek	Wilmington, DE	No data	4.8×10^{-5}	No data	No data
Humbolt Bay[b]	Eureka, CA	No data	8.8×10^{-3}	No data	3.2×10^{-5}
James A. Fitzpatrick	Syracuse, NY	No data	4.1×10^{-5}	No data	6.6×10^{-6}
LaCrosse[b]	LaCrosse, WI	3.2×10^{-4}	0.010	No data	1.0×10^{-4}
LaSalle	Ottawa, IL	No data	No data	No data	No data
Limerick	Philadelphia, PA	2.0×10^{-3}	6.0×10^{-3}	No data	No data

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993 (*continued*)

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Boiling water reactors (*cont.*)					
Millstone	New London, CT	0.066	0.224	6.9×10^{-6}	8.9×10^{-5}
Monticello	St. Cloud, MN	No data	No data	No data	4.8×10^{-4}
Nine Mile Point	Oswego, NY	No data	No data	No data	1.9×10^{-5}
Oyster Creek	Toms River, NJ	No data	No data	No data	6.5×10^{-5}
Peach Bottom	Lancaster, PA	5.2×10^{-4}	1.4×10^{-3}	6.1×10^{-5}	3.0×10^{-4}
Perry	Painesville, OH	8.8×10^{-5}	1.6×10^{-4}	No data	No data
Pilgram	Boston, MA	No data	9.8×10^{-4}	2.4×10^{-5}	7.3×10^{-5}
Quad-Cites	Moline, IL	No data	3.6×10^{-3}	No data	1.8×10^{-4}
River Bend	Baton Rouge, LA	2.6×10^{-4}	2.2×10^{-3}	No data	No data
Shoreham	Brookhaven, NY	No data	No data	No data	No data
Susquehanna	Berwick, PA	No data	2.9×10^{-5}	No data	No data
Vermont Yankee	Brattleboro, VT	No data	No data	No data	9.9×10^{-5}
WNP-2	Richland, WA	6.3×10^{-3}	0.019	6.7×10^{-6}	7.2×10^{-6}
Total		**0.12**	**0.58**	**4.5×10^{-4}**	**3.3×10^{-3}**

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993 (*continued*)

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Pressurized water reactors					
Arkansas One	Russellville, AR	0.054	0.11	No data	3.5×10^{-8}
Beaver Valley	Shippingport, PA	1.2×10^{-3}	2.3×10^{-3}	No data	No data
Big Rock Point	Charlevoix, MI	9.5×10^{-4}	0.012	1.1×10^{-5}	1.5×10^{-4}
Braidwood	Joliet, IL	6.5×10^{-4}	1.7×10^{-3}	No data	No data
Byron	Byron, IL	5.6×10^{-3}	1.5×10^{-3}	No data	No data
Callaway	Fulton, MO	5.3×10^{-4}	7.8×10^{-4}	No data	No data
Calvert Cliffs	Washington, DC	0.14	0.21	3.4×10^{-3}	4.2×10^{-3}
Catawba	Rock Hill, SC	1.6×10^{-3}	3.9×10^{-3}	No data	No data
Comanche Peak	Glen Rose, TX	8.7×10^{-3}	8.7×10^{-3}	No data	No data
Crystal River	Tampa, FL	2.7×10^{-3}	9.3×10^{-3}	No data	3.1×10^{-6}
Davis-Besse	Toledo, OH	1.4×10^{-3}	6.2×10^{-3}	No data	No data
Diablo Canyon	San Luis Obispo, CA	0.036	0.064	3.3×10^{-5}	2.4×10^{-4}
Donald C. Cook	St. Joseph, MI	1.3×10^{-3}	6.3×10^{-3}	No data	7.3×10^{-7}
Fort Calhoun	Omaha, NE	1.3×10^{-3}	0.012	No data	1.7×10^{-6}
H.B. Robinson	Hartsville, SC	3.6×10^{-4}	4.1×10^{-4}	5.8×10^{-6}	1.8×10^{-5}
Haddam Neck	Middletown, CT	0.013	0.023	8.0×10^{-4}	8.8×10^{-3}

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993 (*continued*)

Installation	Location[a]	Annual total site environmental releases for 1993			
		Water		Air	
		^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Pressurized water reactors (*cont.*)					
Harris	Raleigh, NC	2.9×10^{-4}	3.9×10^{-4}	No data	No data
Indian Point[b]	Peekskill, NY	3.0×10^{-3}	0.10	No data	7.0×10^{-4}
Joseph M. Farley	Dothan, AL	1.5×10^{-3}	3.6×10^{-3}	No data	No data
Kewaunee	Green Bay, WI	No data	2.6×10^{-6}	No data	2.0×10^{-6}
Maine Yankee	Wicassett, ME	1.4×10^{-3}	8.2×10^{-3}	No data	4.2×10^{-5}
McGuire	Charlotte, NC	1.6×10^{-3}	4.6×10^{-3}	No data	1.3×10^{-6}
North Anna	NW Richmond, VA	6.2×10^{-3}	9.2×10^{-3}	3.4×10^{-6}	8.2×10^{-5}
Oconee	Greenville, SC	2.7×10^{-3}	0.010	No data	3.9×10^{-4}
Palisades	South Haven, MI	2.4×10^{-4}	4.3×10^{-3}	No data	1.1×10^{-5}
Palo Verde	Phoenix, AZ	No data	No data	2.0×10^{-3}	1.7×10^{-3}
Point Beach	Manitowoc, WI	0.019	0.027	6.9×10^{-3}	6.9×10^{-3}
Prairie Island	Minneapolis, MN	2.8×10^{-3}	3.8×10^{-3}	1.5×10^{-5}	1.8×10^{-5}
R.E. Ginna	Rochester, NY	0.042	0.041	No data	5.2×10^{-6}
Rancho Seco[b]	Sacramento, CA	1.9×10^{-5}	3.6×10^{-4}	No data	No data
Salem	Wilmington, DE	0.81	1.0	No data	7.0×10^{-7}
San Onofre[b]	San Clemente, CA	0.49	0.57	2.5×10^{-5}	4.0×10^{-5}
Seabrook	Portsmouth, NH	No data	3.3×10^{-5}	No data	No data

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993 (*continued*)

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Pressurized water reactors (*cont*.)					
Sequoyah	Daisy, TN	0.086	0.14	No data	No data
South Texas	Bay City, TX	3.4×10^{-3}	5.4×10^{-3}	No data	No data
St. Lucie	Ft. Pierce, FL	0.055	0.083	9.2×10^{-6}	2.1×10^{-5}
Summer	Columbia, SC	1.6×10^{-3}	3.3×10^{-3}	2.2×10^{-5}	2.9×10^{-5}
Surry	Newport News, VA	7.6×10^{-5}	0.011	No data	7.2×10^{-5}
Three Mile Island[b]	Harrisburg, PA	0.026	0.030	1.2×10^{-7}	4.7×10^{-6}
Trojan[b]	Portland, OR	1.0×10^{-3}	4.0×10^{-3}	No data	No data
Turkey Point	Florida City, FL	9.4×10^{-4}	5.5×10^{-3}	No data	9.4×10^{-7}
Vogtle	Augusta, GA	5.6×10^{-3}	7.3×10^{-3}	No data	No data
Waterford	New Orleans, LA	0.013	0.016	No data	No data
Wolf Creek	Burlington, KS	0.022	0.024	No data	No data
Yankee Rowe[b]	Greenfield, MA	4.3×10^{-6}	6.0×10^{-5}	No data	1.0×10^{-7}
Zion	Waukegan, IL	0.014	0.029	2.4×10^{-4}	2.9×10^{-4}
Total		**1.88**	**2.85**	**0.013**	**0.023**

[a]Post office state abbreviations used.
[b]Facilities that are permanently or indefinitely shut down.

6.2.1 Air

Stable cesium is introduced into the atmosphere by resuspension of soil, accidental release from mining and milling pollucite, and emissions from hazardous waste incinerators or coal burning plants. These emissions are expected to be low since cesium occurs naturally in the earth's crust at low concentrations and only small amounts of pollucite are mined annually. Cesium was detected at concentrations of 10.8 and 6.11 mg/m^3 in the effluent of a coal-burning power plant in the western United States (Ondov et al. 1989) and has been identified in the fly ash from municipal incinerators (Mumma et al. 1990, 1991). Fly ash from five municipal waste incinerators in the United States contained cesium at concentrations of 2,100–12,000 ppm (EPA 1990a). Stable cesium has been identified in air at 2 of the 10 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001).

Radioactive isotopes of cesium such as ^{137}Cs and ^{134}Cs have been released to the atmosphere from atmospheric nuclear weapons testing, accidents at nuclear reactors, and nuclear-powered satellites burning up in the atmosphere upon re-entry. The total amount of ^{137}Cs released from weapons testing through 1980 was estimated as $2.6x10^7$ Ci ($9.6x10^{17}$ Bq), 76% of which was released in the northern hemisphere and 24% in the southern hemisphere (WHO 1983). On April 26, 1986, a steam buildup caused an explosion and fire at a nuclear power generating plant in Chernobyl, Russia, releasing an estimated $5.4x10^5$ Ci ($2.0x10^{16}$ Bq) of ^{134}Cs and $1.1x10^6$ Ci ($4.0x10^{16}$ Bq) of ^{137}Cs into the atmosphere over Europe (Watson 1987). Long-range transport spread the radionuclides throughout the Northern Hemisphere. No airborne activity from Chernobyl has been reported south of the equator (Eisler 1995). By early May 1986, these radionuclides were readily detectable in environmental samples collected in North America (Huda et al. 1988). More recent estimates have put the total activity of ^{137}Cs released from the Chernobyl power plant as $2.3x10^6$ Ci ($8.5x10^{16}$ Bq) and $1.2x10^6$ Ci ($4.4x10^{16}$ Bq) for ^{134}Cs (Buzulukov and Dobrynin 1993). On January 24, 1978, the Soviet nuclear-powered satellite Cosmos 954 re-entered earth's atmosphere over the Canadian Arctic, releasing an estimated 86 Ci of ^{137}Cs (Barrie et al. 1992). In October 1957, an accident at the Windscale nuclear weapons plant at Sellafield in the United Kingdom resulted in the release of 595 Ci of ^{137}Cs (ATSDR 1999). Routine activities at nuclear power plants and fuel-reprocessing stations also release ^{137}Cs and ^{134}Cs to the environment on a regular basis. Radiocesium released in airborne effluents from the normal operation of nuclear power plants is considered low in comparison to releases from atmospheric weapons testing and the major releases following accidents at nuclear power plants. In 1998, it was reported that $1.3x10^{-4}$ Ci of ^{134}Cs and $5.1x10^{-3}$ Ci of ^{137}Cs were released to the atmosphere from the Savannah River plutonium processing site in South Carolina (DOE 1998b). In 1993, the Nuclear Regulatory Commission (NRC) estimated that 0.013 Ci of ^{134}Cs and

0.023 Ci of ^{137}Cs were released in airborne effluents from 30 PWR nuclear power plants operating in the United States (NRC 1993b). It was also estimated that $4.6x10^{-4}$ Ci of ^{134}Cs and $3.3x10^{-3}$ Ci of ^{137}Cs were released in airborne effluents from 28 BWR nuclear power plants (NRC 1993b). The total airborne and liquid releases of ^{134}Cs and ^{137}Cs from the individual nuclear power plants are summarized in Table 6-1.

Radioactive ^{134}Cs was not identified in air at the 3 NPL hazardous waste sites where it was detected in some environmental media, but ^{137}Cs was identified in air at 5 of the 22 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001).

6.2.2 Water

Cesium can be released to water surfaces during the mining, milling, and production process of pollucite ore. The natural erosion and weathering of rocks will also lead to cesium's introduction into ground and surface water. Stable cesium was identified in groundwater at 4 sites and surface water at 1 of the 10 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001).

The dumping of high and low level radioactive wastes into the Arctic waters by the former Soviet Union has also led to the release of ^{137}Cs and ^{134}Cs as well as other radioactive nuclides into these waters. In the past, the majority of radioactive cesium released to water surfaces in North America arose from deposition following atmospheric nuclear weapons testing conducted by the United States, primarily during the 1960s (Robbins et al. 1990). Radioactive cesium can be introduced to water from nuclear power plants (accidents and normal operation) and at facilities that produce weapons grade plutonium and uranium. During the period of 1961–1973, it was estimated that about 514 Ci of ^{137}Cs was emitted to the Savannah River watershed due to the activities at the Savannah River plutonium processing plant (Olsen et al. 1989). It was further noted that about 18% of this total (92 Ci) drained directly into the Savannah River (Olsen et al. 1989). In 1998, it was reported that $1.0x10^{-4}$ Ci of ^{134}Cs and 0.19 Ci of ^{137}Cs were released in liquid effluents from the Savannah River plutonium processing site in South Carolina (DOE 1998b). In 1993, the NRC estimated that 1.88 Ci of ^{134}Cs and 2.85 Ci of ^{137}Cs were released in liquid effluents from 30 PWR nuclear power plants operating in the United States (NRC 1993a). It was also estimated that 0.12 Ci of ^{134}Cs and 0.58 Ci of ^{137}Cs were released in liquid effluents from 28 BWR nuclear power plants (NRC 1993a). The EPA reported that the total on-site liquid discharges of ^{137}Cs from containment ponds at the Nevada Test Site was 0.0017 Ci in 1997 (EPA 1999c). It was estimated that 1,622 Ci of ^{137}Cs and 811 Ci of ^{134}Cs were released to the cooling pond surrounding the Chernobyl nuclear power plant following the accident in 1986 (UNSCEAR 1996).

Radioactive ^{134}Cs was not identified in any water samples at the 3 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001). However, ^{137}Cs was identified in groundwater at 4 sites and surface water at 3 of the 22 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001).

6.2.3 Soil

Anthropogenic sources of ^{133}Cs releases to soils include the mining, milling, and processing of pollucite ore. It is also found in the ash of coal burning power plants and municipal waste incinerators. Stable cesium was detected at concentrations of 0.44–2.01 ppm in the bottom ash of municipal solid waste incinerators operating in the United States (Mumma et al. 1990) and at concentrations of 3–23 ppm from a municipal waste incinerator operating in Barcelona, Spain (Fernandez et al. 1992). Stable cesium was identified in soil at 1 site and sediment at 1 of the 10 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001).

Radioactive cesium has been released to soil surfaces by underground nuclear weapons testing, fallout from the accident at the Chernobyl nuclear power plant and fallout from atmospheric weapons testing. About 1,400 underground tests have been performed worldwide, with a total explosive yield of about 90 megatons (ATSDR 1999). Small amounts of ^{137}Cs and ^{134}Cs are also released to soil from the normal operation of nuclear power plants and the storage of spent fuel rods. Not including the 30-km exclusionary zone, an area of approximately $2.4x10^4$ km^2 near the Chernobyl nuclear power plant was contaminated with ^{137}Cs at a deposition density $>5.4x10^{-5}$ Ci/m^2 following the accident in 1986 (UNSCEAR 1996). Within the exclusionary zone the contamination density may have been 2 orders of magnitude greater in limited areas (UNSCEAR 1996). The mean deposition density of ^{137}Cs and ^{134}Cs in four different soils in Devoke, United Kingdom for May 1986 were reported as $3.7x10^{-7}$–$5.4x10^{-7}$ Ci/m^2 and $1.0x10^{-7}$–$1.8x10^{-7}$ Ci/m^2, respectively (Hilton et al. 1993). The concentrations of ^{137}Cs in eight sediment cores of the Danube River, Austria were about 540 pCi/kg in April 1985, but increased to approximately 27,000–81,000 pCi/kg in October 1986, following the accident at the Chernobyl nuclear power plant (Rank et al. 1990). The deposition of ^{137}Cs attributed to the accident at the Chernobyl nuclear power plant in sediment at five different sites in Lake Constance, Germany ranged from $2.7x10^{-7}$ to $2.1x10^{-6}$ Ci/m^2, while the concentration attributed to fallout from nuclear weapons testing since 1963 ranged from $1.4x10^{-7}$ to $5.4x10^{-7}$ Ci/m^2 (Richter et al. 1993). It was estimated that 2,973 Ci of ^{137}Cs and 1,622 Ci of ^{134}Cs were released to the sediments in the cooling pond surrounding the Chernobyl nuclear power plant following the accident in 1986 (UNSCEAR 1996). The deposition density of ^{137}Cs in

123 soil cores collected at the Idaho National Engineering and Environmental Laboratory (INEEL), a site for stored transuranic waste in the United States, ranged from $1.6x10^{-8}$ to $3.4x10^{-7}$ Ci/m^2 (DOE 1998a). The deposition density of ^{137}Cs in soils from Idaho, Montana, and Wyoming ranged from $3.0x10^{-9}$ to $1.1x10^{-7}$ Ci/m^2, and it was assumed that its origin was fallout from the Nevada Test Site (DOE 1998a). The mean deposition density of ^{137}Cs in the top layer (0–8 cm) of soils near the Chernobyl nuclear power plant in 1988 was $8.6x10^{-5}$ Ci/m^2 and the mean deposition density of ^{134}Cs was $1.9x10^{-5}$ Ci/m^2 (Mikhaylovskaya et al. 1993).

It was reported that ^{134}Cs was identified in soil at 1 site, but was not detected in any sediment samples at the 3 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001). It was also reported that ^{137}Cs was identified in soil at 13 sites and sediment at 5 of the 22 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

The transport and partitioning of particulate matter in the atmosphere is largely dependent upon the physical properties of the matter such as size and density as well as the meteorological conditions such as temperature, the microphysical structure of the clouds, and rainfall rate. The particle size of ^{137}Cs released to the atmosphere following the Chernobyl nuclear accident was in the range of 0.1–10 μm, (Hirose et al. 1993). Particles <5 μm in diameter usually have low deposition velocities and are transported long distances before being removed from the atmosphere. Atmospheric cesium is deposited on land and water via wet and dry deposition and the deposited cesium may be re-suspended to the atmosphere by disturbances that occur on the ground such as vehicular traffic and construction activity. The wet deposition velocity of ^{137}Cs at Tsukauba, Japan from May 5 to May 30, 1986 ranged from 0.0026 to 0.110 m/second, and the largest value recorded was during a period of heavy rainfall (Hirose et al. 1993). The mean deposition velocity (wet and dry) of ^{137}Cs measured in Prague, Czechoslovakia was reported as 0.08 m/second from 1989–1992 and the mean flux rate was 1,108 pCi/m^2-year (Rybacek et al. 1994).

Since cesium does not volatilize from water, transport of cesium from water to the atmosphere is not considered likely, except by windblown sea sprays. Most of the cesium released to water will adsorb to suspended solids in the water column and ultimately be deposited in the sediment core. Cesium can also

bioconcentrate and has been shown to bioaccumulate in both terrestrial and aquatic food chains. Mean bioconcentration factors (BCF) for ^{137}Cs of 146, 124, and 63 were reported for fish, brown macroalgae, and molluscs, respectively (Fisher et al. 1999). Mean BCF values of 92, 58, 39, and 150 were reported for ^{137}Cs in cod, haddock, plaice, and whiting, respectively (Steele 1990). In a study of aquatic organisms inhabiting the Ottawa River, a 4-fold increase of ^{137}Cs levels was observed with each trophic level (Rowan et al. 1998). The levels of ^{137}Cs in lake trout from Great Slave Lake, Canada were consistently higher than levels found in food sources and a biomagnification factor of 1.9 was calculated for lake trout, relative to their food sources. The biomagnification factor was 3.5 for large mature trout populating the lake (Rowan et al. 1998). It was shown that the bioconcentration and bioaccumulation of ^{137}Cs by aquatic organisms is significantly reduced in waters with a large humic content and high levels of potassium cations (Penttila et al. 1993). Because of the high potassium concentration in oceans, the transfer of ^{137}Cs and ^{134}Cs to fish is much greater in freshwater and the activity of freshwater fish may be 100 times that of ocean fish, given the same cesium concentration in the water (WHO 1983).

In soil surfaces, cesium has low mobility in comparison to other metals and usually does not migrate below a depth of 40 cm. The majority portion of cesium is retained in the upper 20 cm of the soil surface (Korobova et al. 1998; Ruse and Peart 2000; Takenaka et al. 1998). Vertical migration patterns of ^{137}Cs in four agricultural soils from southern Chile indicated that approximately 90% of the applied cesium was retained in the top 40 cm of soil, and that in one soil, essentially 100% was bound in the upper 10 cm (Schuller et al. 1997). Migration rates of radiocesium were derived from the depth distribution profiles and were in the range of 0.11 to 0.29 cm/year (Schuller et al. 1997). The vertical migration patterns of ^{90}Sr and ^{137}Cs produced from the atomic bomb exploded in Nagasaki, Japan were studied over a 40-year period (Mahara 1993). Over this period, 95% of the cesium remained in the top 10 cm of the soil surface and no cesium was detected below a depth of 40 cm. In contrast, only 70% of ^{90}Sr was located within a depth of 10 cm and a small percentage was detectable below a depth of 200 cm. The *in situ* vertical migration rate of ^{90}Sr was calculated as 0.42 cm/year and the migration rate of ^{137}Cs was 0.10 cm/year (Mahara 1993). Soil adsorption coefficients (K_d) of five radionuclides (^{54}Mn, ^{60}Co, ^{65}Zn, ^{85}Sr, and ^{137}Cs) were measured for 36 agricultural soils collected in Japan. It was determined that ^{137}Cs had the largest median K_d of all five radionuclides, and that a positive correlation was observed between the adsorption coefficient and exchangeable potassium content in the soil (Yasuda et al. 1995). No correlations were observed for other soil properties such as pH, water content, cation exchange capacity, and exchangeable calcium. Other studies have reported that clay and zeolite minerals strongly bind cesium cations and can therefore reduce the bioavailability of cesium and the uptake in plants by irreversibly binding cesium in interlayer positions of the clay particles (Paasikallio 1999). Experiments conducted by growing plants in a peat soil showed that the introduction of zeolites into the soil-plant system decreased the uptake of ^{134}Cs

in plants by a factor of 8 (Shenber and Johanson 1992). The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays and zeolites (Hakem et al. 1997). Soils rich in organic matter adsorb cesium, but the cesium adsorbed in the organic fraction is readily exchangeable and highly available for plant uptake (Sanchez et al. 1999). Regions in Venezuela, Brazil, and Russia have been identified where a great deal of rain is encountered, the soil is peaty or podzolic (a type of forest soil characterized by high leachability) , and the mobility of cesium is considerably greater than in other soils (LaBrecque and Rosales 1996; WHO 1983).

The plant/soil concentration ratio (activity/kg of plant/activity/kg of soil) of ^{137}Cs for field crops in southern Finland ranged from 0.01 to 0.26. In northern Finland, this ratio ranged from 0.01 to 2.29, with the lowest values occurring in clay and silt soils (Paasikallio et al. 1994). The plant/soil concentration ratios for a series of vegetables and grains decreased in the following order: lettuce, cabbage>carrot, potato>cereals, onion; for fruits, the order was: blackcurrant>strawberry>apple (Paasikallio et al. 1994). The mean plant/soil concentration ratios of ^{137}Cs for trees at the Hanford Waste Site in the United States were 0.03 (roots), 0.06 (cores), and 0.02 (leaf/twig) (Landeen and Mitchell 1986).

6.3.2 Transformation and Degradation

6.3.2.1 Air

When pure cesium metal is exposed to air, an explosion-like oxidation occurs, forming a mixture of cesium oxides (Cs_2O, Cs_2O_2, and Cs_2O_3). Cesium compounds released to the atmosphere will eventually settle to earth by wet and dry deposition. Radioactive forms of cesium such as ^{137}Cs and ^{134}Cs are continuously transformed to stable isotopes of barium or xenon by the natural process of radioactive decay. The pathways and mechanisms of these reactions have been described in Chapter 4.

6.3.2.2 Water

When pure cesium metal is released to water, a vigorous reaction occurs yielding cesium hydroxide (CsOH), the strongest base known, and hydrogen gas, which may ignite spontaneously. In general, cesium compounds are very water soluble, and exist primarily as the Cs^+ cation. Under normal environmental conditions, Cs^+ cations are neither degraded nor transformed, but may adsorb to suspended solids and sediment in the water column, forming insoluble complexes.

6.3.2.3 Sediment and Soil

Cesium salts and most cesium compounds are generally very water soluble, with the exception of cesium alkyl and aryl compounds, which have low water solubility. Cesium cations have a low hydration energy and can react with clay minerals, zeolites, or soils with a high percentage of exchangeable potassium, forming insoluble, immobile complexes.

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to cesium depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Radioactive cesium is measured in units of activity, not mass. A great deal of monitoring data for radioactive cesium in environmental samples prior to and shortly following the accident at the Chernobyl nuclear power plant on April 26, 1986 have been included.

6.4.1 Air

Data reporting the background levels of ^{133}Cs in the atmosphere are limited. Since the quantity of cesium mined and milled is small, it is expected that background concentrations in the atmosphere will be low. The concentration of ^{133}Cs measured in the South Pole during 1974–1975 was reported in the range of 0.072 to 0.14 pg/m^3, with a mean of 0.10 pg/m^3 (Maenhaut et al. 1979). The maximum airborne concentrations of ^{133}Cs at the Tera Nova Bay Station in the Arctic were reported as 20–90 pg/m^3 during the 1989–1990 Italian expedition and 10–60 pg/m^3 for the 1990–1991 expedition (Chiavarini et al. 1994). The average concentration of ^{133}Cs in precipitation collected in Lennox, Massachusetts during the summer of 1984 was reported as 0.0075 µg/L, with a range of 0.0050 to 0.024 µg/L (Dasch and Wolff 1989).

Radioactive ^{134}Cs and ^{137}Cs have been detected at various concentrations (activities) in the atmosphere following the accident at the Chernobyl nuclear power plant on April 26, 1986. The greatest concentrations were observed in locations throughout Russia and Europe, but detectable levels were measured globally, including in North America. The concentrations of ^{134}Cs in the air above the destroyed reactor were 1,756.8 pCi/m^3 on May 8 and 54.0 pCi/m^3 on May 18 (Buzulukov and Dobrynin 1993). The concentrations of ^{137}Cs in the air above the destroyed reactor were 8,918.9 pCi/m^3 on May 8 and 135.1 pCi/m^3 on May 18 (Buzulukov and Dobrynin 1993). Atmospheric concentrations of ^{134}Cs in Belgrade, Yugoslavia were 5.4x10^{-4} (May 1, 1986), 160.5 (May 2, 1986), 145 (May 3, 1986), and

25.4 (May 4, 1986) pCi/m^3, while atmospheric concentrations of ^{137}Cs were 0.001 (May 1, 1986), 324 (May 2, 1986), 276 (May 3, 1986), and 49 (May 4, 1986) pCi/m^3 (Ajdacic and Martic 1990). These concentrations decreased significantly over time as advection, wet deposition, and dry deposition lowered the atmospheric concentrations. The average monthly atmospheric concentration of ^{137}Cs in Belgrade that were measured during 1991–1996 ranged from $1.3x10^{-4}$ to $2.0x10^{-3}$ pCi/m^3, with a pronounced maximum during the winter months (Todorovic et al. 1999). Monitoring data from Prague, Czechoslovakia indicated that there was a gradual decrease in the atmospheric concentration of ^{137}Cs from May 1986 to February 1992. Maximum concentrations were measured immediately following the accident at the Chernobyl nuclear power plant (approximately 0.05 pCi/m^3) and gradually decreased over a 6-year period (Rybacek et al. 1994). The atmospheric concentration of ^{134}Cs decreased more rapidly, presumably due to the shorter half-life of ^{134}Cs. The concentration of ^{137}Cs in the atmosphere of Thessaloniki, Greece ranged from $8.1x10^{-4}$ to 0.044 pCi/m^3 from July 1987 to December 1988, and the concentration of ^{137}Cs in rainfall ranged from 0.27 to 34 pCi/L from November 1986 to February 1989 (Papastefanou et al. 1989). During a heavy rainfall event occurring on May 6, 1986, ^{137}Cs was detected at a concentration of 46,000 pCi/L (Papastefanou et al. 1989). The concentration of ^{137}Cs in Tsukuba, Japan during May 1986 ranged from about 0.054 to 1.6 pCi/m^3 (Hirose et al. 1993). The average atmospheric concentrations of ^{134}Cs and ^{137}Cs in eastern Canada were reported as 0.024 and 0.046 pCi/m^3, respectively, during May 10–24, 1986 (Huda et al. 1988). The maximum atmospheric concentration of ^{137}Cs measured in New York City in May 1986 was reported as 0.26 pCi/m^3 (Feely et al. 1988). The average concentrations of ^{137}Cs and ^{134}Cs in Barrow, Alaska were reported as 0.027 and 0.019 pCi/m^3, respectively, for the month of May 1986 (DOE 1986). In 1975, the maximum concentration of ^{137}Cs in the atmosphere, in Poland was 1.89 pCi/m^3 (Glowiak et al. 1977b).

6.4.2 Water

The background concentration of ^{133}Cs in fresh water lakes and rivers is ordinarily in the range of 0.01 to 1.2 µg/L, and the concentration in seawater is about 0.5 µg/L (WHO 1983). Stable cesium was detected in streams that feed into the Tamagawa River, Japan at concentrations of $9x10^{-4}$ to 0.093 µg/L (Tanizaki et al. 1992). Studies from five distinct geochemical areas of the semi-arid endorheic region of the Famatina Range (La Rioja, Argentina) have shown some areas contain high levels of ^{133}Cs in natural waters and sediment (Fernandez-Turiel et al. 1995). The cesium concentration in fresh water systems of this region ranged from 0.58 to 3.69 µg/L (Fernandez-Turiel et al. 1995). The concentrations of 19 trace metals were studied in drinking water and groundwater supplies in southern Nigeria. Stable cesium was detected in groundwater and drinking water at mean concentrations of 0.61 µg/L (range 0.09–3.72 µg/L)

and 0.35 µg/L (range 0.05–4.32 µg/L), respectively (Asubiojo et al. 1997). It was further noted that the mean concentration of cesium in drinking water was lower than the mean concentration of any of the other trace elements analyzed.

High and low level radioactive wastes have been dumped by the former Soviet Union into remote Arctic waters, leading to the release of radioactive cesium into the Kara and Barents Seas. The level of ^{137}Cs in surface water of the Barents Sea and Kara Sea was 0.14 and 0.16 pCi/L, respectively, and it was also detected in deep water of the Barents Sea at a concentration of 0.15 pCi/L (Fisher et al. 1999). The concentration of ^{137}Cs in the Black Sea was in the range of 2.7 to 8.1 pCi/L for the period of 1991–1996, with the exception of the spring of 1992, when concentrations as high as 43 pCi/L were observed (Strezov et al. 1999). From 1988 to 1991, the mean concentrations of ^{137}Cs and ^{134}Cs along the Spanish coast of the Mediterranean Sea were 0.13 and 0.0072 pCi/L, respectively (Molero et al. 1999). Due to its shorter half-life, ^{134}Cs was detected in all 14 samples collected in 1988 and 1989, but only in 3 samples collected in 1990 and 1991, suggesting that the ^{134}Cs levels observed in surface Mediterranean waters during this period were due exclusively to Chernobyl-related deposition. The ^{137}Cs concentration incorporated into the Mediterranean Sea near the Spanish coast from the post-Chernobyl fallout was about 0.032 pCi/L, which was approximately a 33% increase over previous levels (Molero et al. 1999). Maximum ^{137}Cs and ^{134}Cs levels in the immediate vicinity of nuclear power plants located on the southern Catalan shore of the Mediterranean were 0.57 and 0.059 pCi/L (Molero et al. 1999). Concentrations of ^{137}Cs in lakes and streams in Devoke, United Kingdom decreased exponentially from a maximum concentration of about 8.1 pCi/L on May 6, 1986 to about 0.027 pCi/L 1,200 days later (Hilton et al. 1993). The mean concentration of ^{137}Cs in six lakes located in central Finland ranged from 111 pCi/L in 1987 to 8.1 pCi/L in 1989 (Penttila et al. 1993).

The concentration of ^{137}Cs and ^{134}Cs in groundwater at 18 U.S. Department of Energy (DOE) facilities was reported in the range of 2.7×10^{-3} to 1.83×10^{3} pCi/L (DOE 1992). The concentration of ^{137}Cs measured in groundwater wells at Carlsbad, New Mexico (the site of Project GNOME) ranged from 99 to 6.8×10^{5} pCi/L in 1997 (EPA 1999c). The concentration of ^{137}Cs in groundwater at the Chernobyl nuclear power plant was in the range of 40.5–1,100 pCi/L in 1988 and 29.7–129.7 pCi/L in 1989 (Prister et al. 1990). The mean concentration of ^{137}Cs in drinking water in Poland in 1974 was reported as 0.2 pCi/L (Glowiak et al. 1977b). These concentrations in water may be compared to the federal radiation safety standards. For continuous ingestion over a lifetime, the maximum concentrations of ^{134}Cs and ^{137}Cs in drinking water are limited to 900 and 1,000 pCi/L respectively. It should be noted however, that these

limits assume no other intake of radioactivity. If other radioisotopes are ingested, then the intake limits for each must be adjusted proportionately (NRC 1999a).

6.4.3 Sediment and Soil

Stable cesium occurs naturally in the earth's crust at low concentrations. Granites contain an average cesium concentration of about 1 ppm and sedimentary rocks contain about 4 ppm (Burt 1993). Others have estimated cesium concentration of granites as high as 5 ppm (WHO 1983). Stable cesium was detected in dust samples from roadside and pedestrian traffic in Nagpur, India at concentrations of 1.53–3.63 µg/g, with the largest value obtained in the vicinity of a metals factory (Chutke et al. 1995). It was reported that ^{133}Cs was detected at concentrations ranging from 0.9 to 2.2 µg/g in alluvial sediments in the Sava River, Croatia (Vertacnik et al. 1997). The concentration range of ^{133}Cs in river sediment from five distinct geochemical areas of the semi-arid endorheic region of the Famatina Range (La Rioja, Argentina) was 2.28–6.20 µg/g (Fernandez-Turiel et al. 1995).

The concentration of ^{137}Cs in soils of Thessaloniki, Greece ranged from 1,440 to 35,324 pCi/kg (average 8,154 pCi/kg) and the concentration of ^{134}Cs ranged from about 270 to 5,676 pCi/kg during the period of August 1986 to February 1989, with most of the fallout attributed to the accident at the Chernobyl nuclear power plant (Papastefanou et al. 1989). The concentration of ^{137}Cs in 10 uncultivated fields from southern England ranged from 0 to 946 pCi/kg, with the highest levels contained in the top 10 cm of the soil surface (Owens et al. 1996). The concentration of ^{137}Cs in five cultivated fields ranged from 0 to 540 pCi/kg, and the concentrations were well distributed from the surface to the plough layer (Owens et al. 1996). The concentration of ^{137}Cs in three soils in Hong Kong receiving a large amount of rainfall ranged from 32 to 201 pCi/kg (Ruse and Peart 2000). The concentration of ^{137}Cs in sediment from the Ribble Estuary, England near the British Nuclear Fuels Laboratory ranged from 270 to 1,351 pCi/kg (Brown et al. 1999). The average concentration of ^{137}Cs in sediment from Lake Ontario was reported as 8,108 pCi/kg, and suspended solids from the Niagara River contained 324 pCi/kg (Platford and Joshi 1989). The average concentrations of ^{137}Cs in uncultivated soils in northern Poland were reported as 616–4,170 pCi/kg from 1988–1991 (Pietrzak-Flis et al. 1994). The mean concentration of ^{137}Cs in surface soil samples from the Los Alamos nuclear laboratory test site during the period of 1974–1996 was reported as 611 pCi/kg (Fresquez et al. 1998). Concentrations of ^{137}Cs around the perimeter of the site and background concentrations off the site were reported as 589 and 419 pCi/kg , respectively. The concentration of ^{137}Cs and ^{134}Cs in soils and sediments at 18 U.S. DOE facilities was reported to range from 20 to 4.69×10^7 pCi/kg (DOE 1992). The concentration of ^{137}Cs in sediment from

the Savannah River ranged from 5 to 100 pCi/kg in 1986, while the concentration in suspended solids and particulate matter ranged from 240 to 4.324×10^6 pCi/kg (Olsen et al. 1989). The mean concentration of ^{137}Cs in soil at the Hanford Site in the United States was 4,540 pCi/kg (Landeen and Mitchell 1986). The mean concentration of ^{137}Cs in soils taken from two high-elevation sites in northern Colorado ranged from 4,054 to 7,027 pCi/kg (Ulsh et al. 2000).

6.4.4 Other Environmental Media

Data regarding the concentrations of ^{137}Cs, ^{134}Cs, and ^{133}Cs in various animals are shown in Table 6-2. Concentrations are dependent on the location, date, and level of exposure. For example, the concentrations of ^{137}Cs in bullhead catfish inhabiting an abandoned nuclear reactor reservoir at the Savannah River site in South Carolina were as high as 1.54×10^5 pCi/kg (McCreedy et al. 1997), but concentrations in various freshwater species of fish in the Ottawa River were in the range of 54 to 351 pCi/kg (Rowan et al. 1998). After the accident at the Chernobyl nuclear power plant, the average concentrations of ^{137}Cs in perch, pike, and roach obtained from 52 freshwater lakes in Finland were 55,811, 66,297, and 15,270 pCi/kg, respectively, in 1988. By contrast, the mean concentrations in 1992 had fallen to 14,324, 18,567, and 5,892 pCi/kg, respectively (Sarkka et al. 1996). The concentration of total ^{137}Cs and ^{134}Cs measured in body tissue from sheep in Ireland from 1989 to 1991 ranged from about 2,700 to 10,811 pCi/kg, with highest levels observed during November of 1989 (McGee et al. 1993).

The concentrations of ^{133}Cs in a series of plants from Britain were studied over a 1-year period. Concentrations of 50–300 ng/g were measured, with the highest levels observed during the summer and fall months. The median concentration of ^{133}Cs in poplar leaves collected in Bulgaria was reported as 75 ng/g, while concentrations in land plants ranged from 30 to 440 ng/g (Djingova et al. 1995). In contrast to the radioactive isotopes of cesium, concentrations of ^{133}Cs depend primarily on the root uptake from soil and not on atmospheric deposition. Lichens and mosses have been shown to trap and retain ^{137}Cs and ^{134}Cs more so than vascular plants, due to their relatively large surface area. Lichens and mosses from northern Greece contained ^{137}Cs levels of 6.6×10^4–5.1×10^5 pCi/kg during the period of 1989–1991 (Papastefanou et al. 1992) and moss samples from Finland collected in 1988–1989 contained 4.3×10^4–9.7×10^5 pCi/kg (Penttila et al. 1993). The mean concentration of ^{137}Cs in three species of lichens collected in August 1986 from Megalopolis, Greece were 2.6×10^4–3.3×10^4 pCi/kg, while the mean concentrations for the same three species of lichens collected in October 1996 had fallen to 3,324–7,892 pCi/kg (Riga-Karandinos and Karandinos 1998). Mushrooms, lichens, and mosses obtained near Manitoba, Canada in August 1986 contained ^{137}Cs at mean concentrations of 6.4×10^5, 8.6×10^4, and

Table 6-2. Concentration of ^{133}Cs, ^{134}Cs, and ^{137}Cs in Animals

Species	Location and date	^{133}Cs (µg/g)	^{134}Cs (Bq/kg)	^{137}Cs (Bq/kg)	Source
Royal albatross (N=3)	Indian Ocean (1994)	0.009–0.025 (0.014 mean); liver	No data	No data	Kim et al. 1998
Black-footed albatross (N=18)	North Pacific (1985)	0.007–0.049 (0.022 mean); liver	No data	No data	Kim et al. 1998
Black-browed albatross (N=9)	Indian Ocean (1994)	0.013–0.042 (0.022 mean); liver	No data	No data	Kim et al. 1998
White-capped albatross (N=3)	Indian Ocean (1994)	0.011–0.039 (0.029 mean); liver	No data	No data	Kim et al. 1998
Yellow-nosed albatross (N=4)	Indian Ocean (1994)	0.0029–0.079 (0.022 mean); liver	No data	No data	Kim et al. 1998
Grey-headed albatross (N=10)	Indian Ocean (1994)	0.011–0.031 (0.016 mean); liver	No data	No data	Kim et al. 1998
Northern giant petrel (N=6)	Indian Ocean (1994)	0.005–0.034 (0.015 mean); liver	No data	No data	Kim et al. 1998
Northern fulmar (N=17)	North Pacific (1985)	0.008–0.036 (0.016 mean); liver	No data	No data	Kim et al. 1998
Bluefin tuna (N=14)	Newfoundland (1990)	0.08–0.24 (0.13 mean); muscle	No data	No data	Hellou et al. 1992a
Cod (N=12)	Newfoundland (1990)	0.16–0.24 (0.19 mean); muscle	No data	No data	Hellou et al. 1992b
Cod (N=10)	Newfoundland (1990)	0.14–0.36 (0.23 mean); muscle	No data	No data	Hellou et al. 1992b
Pilot whale (N=9)	North Atlantic (1987–1996)	0–0.010 (0.006 mean); liver	No data	No data	Becker et al. 1997
White-sided dolphin (N=4)	North Atlantic (1987–1996)	0.027–0.042 (0.032 mean); liver	No data	No data	Becker et al. 1997

Table 6-2. Concentration of ^{133}Cs, ^{134}Cs, and ^{137}Cs in Animals (*continued*)

Species	Location and date	^{133}Cs (µg/g)	^{134}Cs (Bq/kg)	^{137}Cs (Bq/kg)	Source
Beluga whale (N=15)	Arctic (1987–1996)	0.021–0.046 (0.031 mean); liver	No data	No data	Becker et al. 1997
Ringed seal (N=13)	Arctic (1987–1996)	0.0045–0.048 (0,020 mean); liver	No data	No data	Becker et al. 1997
Woodcock (N=24)	Ireland (1986)	No data	3.9–206.4; muscle	6.2–565.5; muscle	Pearce 1995
Duck (N=5)	Ireland (1986)	No data	2.2–14.3; muscle	6.4–18.0; muscle	Pearce 1995
Snipe (N=5)	Ireland (1986–1987)	No data	1.0–5.4; muscle	3.6–16.9; muscle	Pearce 1995
Reindeer (N=8)	Northern Sweden (1986–1987)	No data	No data	900 (mean); muscle	Ahman and Ahman 1994
Deer (N=11)	Los Alamos (1991–1998)	No data	No data	2,516 (mean); muscle	Fresquez et al. 1999a
Deer (N=11)	Los Alamos (1991–1998)	No data	No data	888 (mean); bone	Fresquez et al. 1999a
Caribou (N=18)	Saskatchewan (1995)	No data	No data	58 (mean); bone	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	228 (mean); liver	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	367 (mean); muscle	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	553 (mean); kidney	Thomas and Gates 1999
Caribou (N=36)	Alaska (1987)	No data	No data	26–232; neck	Allaye-Chan et al. 1990
Caribou (N=36)	Alaska (1987)	No data	No data	28.4–201.1; shoulder	Allaye-Chan et al. 1990
Caribou (N=36)	Alaska (1987)	No data	No data	30.2–166.5; back	Allaye-Chan et al. 1990

$8.4x10^4$ pCi/kg, respectively (Mihok et al. 1989). Since caribou and reindeer consume large amounts of this vegetation during the winter months, high levels of ^{137}Cs and ^{134}Cs have been detected in these animals.

Stable cesium has been detected infrequently in food products at low concentrations. The average concentration of ^{133}Cs in 110 onion samples collected in Denmark was 0.21 µg/kg, with a range of not detected to 0.98 µg/kg (Bibak et al. 1998). By comparison, other elements such as calcium and potassium had mean concentrations of $2x10^5$ and $1.6x10^6$ µg/kg, respectively. The concentration range of ^{133}Cs in wheat flour samples collected in Pakistan was 6.7–11.2 ppb, but ^{133}Cs was not detected from wheat flour samples from America (Ahman et al. 1994).

Levels of ^{137}Cs were below detection limits for all foods analyzed for in the U.S. Food and Drug Administration (FDA) Total Diet Study in 1991–1996 with the exception of honey (Capar and Cunningham 2000). The concentration of ^{137}Cs in honey from the 1995 Market Basket Survey was 6.7 Bq/kg (181.1 pCi/kg), which is almost 200 times lower than the regulatory level of ^{137}Cs in foods. The average concentrations of total ^{137}Cs and ^{134}Cs in milk powder, infant milk powder, infant cereal, meat, lentil, wheat, and macaroni samples from Saudi Arabia were 514, 351, 486, 162, 270, 676, and 351 pCi/kg, respectively (Abdul-Majid et al. 1992). For the month of June 1986, the average concentrations of total ^{137}Cs and ^{134}Cs in milk, green vegetables, fruit, lamb, and beef were reported as 3,243, 2,703, 2,703, 8,108, and 1,622 pCi/kg in high deposition areas of the United Kingdom (Cumbria, north Wales, Scotland, northern Ireland, and the Isle of Man) (Mondon and Walters 1990). It was also estimated that the concentration of total ^{137}Cs and ^{134}Cs was <676 pCi/kg in each of these food sources in areas of low deposition during this time frame. The maximum concentration of ^{137}Cs in pasteurized milk from 65 cities in the United States was 14 pCi/L in May 1989 (EPA 1989). The concentration of ^{137}Cs in fresh milk from Chester, New York and pasteurized milk samples from New York City in May 1986 ranged from about 5.4 to 18.9 pCi/L (Feely et al. 1988). Using radiological surveys from 1978 and 1985–1986, the concentration of ^{137}Cs in 44 adult food groups from the Rongelap Island and Rongelap Atoll was in the range of 0.52–13,000 pCi/kg (Robinson and Phillips 1989).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

General population exposure to cesium occurs by ingestion of food and drinking water, by inhalation of ambient air, and dermal contact with cesium in soil. Since ^{133}Cs is found in low concentrations in the environment, the exposure to the general population is expected to be low. The National Health And Nutritional Examination Survey (NHANES), conducted by the Centers for Disease Control (CDC), reported that the geometric mean concentration of ^{133}Cs in the urine of 1,006 U.S. residents was 4.7 µg/L in 1999 (CDC 2001). Since the sample size of the 1999 survey was small and was conducted in only 12 locations across the United States, the CDC will combine 1999 and year 2000 data and publish an updated report in the future. Occupational exposure to ^{133}Cs occurs primarily through inhalation and dermal contact at workplaces where pollucite is mined or cesium compounds are manufactured or used. The National Occupational Exposure Survey (NOES) conducted by the National Institute for Occupational Safety and Health (NIOSH) from 1981 to 1983 has estimated that 16,461 workers (4,276 of these were female) were potentially exposed to cesium and cesium compounds in the United States (NOES 1989). The NOES database does not contain information on the frequency, level, or duration of the exposure of workers to any of the chemicals listed therein. They are surveys that only provide estimates of workers potentially exposed to the chemicals. The median concentration of ^{133}Cs in the lungs of metal workers in northern Sweden was 0.016 µg/g and was lower than the median concentration of 0.021 µg/g for a control group that was not occupationally exposed (Hewitt 1988). The range of ^{133}Cs concentrations in lung tissue of coal miners from the United Kingdom was 0.07–0.91 µg/g (Hewitt 1988).

Exposure to radioactive cesium is more important from a health perspective than exposure to stable cesium. The primary source of radioactive cesium in the environment is due to fallout from past atmospheric nuclear weapons tests and the Chernobyl accident. Additional contributions from the normal operation of nuclear power plants and other nuclear facilities are small by comparison. Current exposure of the general U.S. population to ^{134}Cs and ^{137}Cs is expected to be low since atmospheric testing of nuclear weapons has been discontinued for many years and Chernobyl-related fallout was low in the United States. As discussed in Appendix A, the average annual effective dose of ionizing radiation (including ^{134}Cs and ^{137}Cs) to the U.S. population from anthropogenic sources are negligible in comparison to natural sources, especially radon and its decay products.

The average daily intake (AVDI) of ^{137}Cs and ^{134}Cs was estimated for adult males residing in the Ukraine in 1994, based upon total diet samples. The mean intake of ^{137}Cs was estimated as 109 pCi/day and the mean intake of ^{134}Cs was estimated as 8.1 pCi/day (Shiraishi et al. 1997). Based on dietary patterns and

the concentration of radiocesium in food sources, the total dietary intakes of ^{134}Cs and ^{137}Cs for typical adults residing in Croatia for the month of May 1986 were estimated as $2.8x10^4$ and $5.9x10^4$ pCi, respectively (Lokobauer et al. 1988). The mean monthly levels of ^{137}Cs in human muscle tissue in Graz, Austria were reported as 1,519 (July 1986 to June 1987), 1,049 (July 1987 to June 1988), 340.5 (July 1988 to June 1989), and 202.7 pCi/kg (July 1989 to June 1990), with a maximum value of 9,584 pCi/kg observed in an individual during September 1986 (Rabitsch et al. 1991). The monthly averages for ^{134}Cs were about half of those reported for ^{137}Cs. By comparison, the maximum concentration of ^{137}Cs in muscle tissue from Harwell, England in 1959 was reported as 224 pCi/kg, the mean concentration in muscle tissue from Massachusetts during January 1961 to June 1962 was 100 pCi/kg, and the mean concentration in human muscle tissue obtained from Japan during April to December 1963 was reported as 119 pCi/kg (Rabitsch et al. 1991). The mean concentrations of ^{137}Cs in the urinary excretion of Italians in northern Italy were 7.3 and 6.2 pCi/day in 1995 and 1996, respectively (Ropolo and Cesana 1997). These values were about two orders of magnitude less than values reported for measurements taken in 1987. The mean concentration of ^{137}Cs in brain, heart, liver, gonads, muscle, bone, and teeth were 0.440, 1.860, 0.490, 2.440, 0.017, 0.106, and 0.23 pCi/g, respectively, for adult cadavers over 34 years of age in Poland during 1975 (Glowiak et al. 1977a). The mean body burdens of ^{137}Cs for adults in Helsinki, Viitasaari and Ammans, Finland from 1987 to 1994 are given in Table 6-3 (Rahola and Suomela 1998).

Persons employed at nuclear power facilities and waste disposal sites are potentially exposed to higher levels of ^{137}Cs and ^{134}Cs than the general population. The NOES conducted by NIOSH from 1981 to 1983 estimated that 13,148 workers (1,294 of these were female) were potentially exposed to ^{134}Cs and ^{137}Cs in the United States (NOES 1989).

6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in 3.7 Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults.

Table 6-3. The Mean Body Burdens of ^{137}Cs for Adults in Finland from 1987–1994[a]

Location	Date	Concentration (pCi/kg)
Helsinki, Finland	1987	649
	1988	568
	1989	405
	1990	297
	1991	246
	1992	214
	1993	195
	1994	181
Viitasaari, Finland	1987	3,514
	1988	1,946
	1989	1,595
	1990	1,081
	1991	838
	1992	676
	1993	649
	1994	514
Ammans, Finland	1987	2,892
	1988	3,108
	1989	2,243
	1990	2,486
	1991	1,568
	1992	1,405
	1993	865
	1994	811

[a]Rahola and Suomela 1998

The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993a).

As for adults in the general population, exposures of children to cesium occur from normal ingestion of food and drinking water, inhaling air, and dermal contact with cesium in soil. No information on cesium levels in amniotic fluid, meconium, cord blood, or neonatal blood was available.

Radioactive cesium was detected in several pasteurized milk and breast milk samples worldwide and since children tend to consume large amounts of milk, this represents an important source of childhood exposure. The maximum concentration of ^{137}Cs in pasteurized milk from 65 cities in the United States was 14 pCi/L in May 1989 (EPA 1989). Concentrations of ^{137}Cs in human milk samples from several U.S. cities from 1956 to 1961 were <20 pCi/L (Eaman 1986). The overall ^{137}Cs concentration in milk taken 7 days postpartum from 37 mothers in two Italian hospitals were 5.8–115 pCi/L (Eaman 1986). In a study of females from northern Sweden, ^{137}Cs was detected in breast milk from 8 out of 12 mothers at concentrations of 7.3–178.4 pCi/kg (Johansson et al. 1998). The infants of these mothers who were breast-fed had whole-body levels of ^{137}Cs in the range of 45.9–675.7 pCi/kg (Johansson et al. 1998). Based on dietary patterns and the concentration of radiocesium in food sources, the total dietary intakes of ^{134}Cs and ^{137}Cs for children (10-years-old) residing in Croatia for May–June 1986 were estimated as 43,000 and 190,000 pCi, respectively (Lokobauer et al. 1988). For infants (1 year of age), it was estimated that the total intakes of ^{134}Cs and ^{137}Cs were 46,000 and 170,000 pCi, respectively (Lokobauer et al. 1988). The total intakes of ^{134}Cs and ^{137}Cs for adults during this same time period were estimated as 40,000 and 84,000 pCi, respectively (Lokobauer et al. 1988). The higher intakes of ^{134}Cs and ^{137}Cs for infants and children were traced to a much greater consumption of contaminated milk.

The tendency of young children to ingest soil, either intentionally through pica or unintentionally through hand-to-mouth activity, is well documented. These behavioral traits can result in ingestion of cesium present in soil and dust. Soil ingestion may be a potentially important exposure pathway in areas that have historically had a great deal of ^{134}Cs and ^{137}Cs deposited onto soil surfaces from the accident at the Chernobyl nuclear power plant or fallout from weapons testing. Playing in contaminated soil could also lead to dermal and external exposure. Ingested cesium is adsorbed strongly to soils and may not be in bioavailable form. A study in which 102 healthy volunteers were fed ^{134}Cs-contaminated soil pellets,

only about 1% of the original amount was absorbed and on average, about 60% of the intake was excreted within 48 hours (LeRoy et al. 1966).

It is unlikely that children whose parents are employed at nuclear power generating plants and facilities that store or handle radioactive waste are exposed to ^{134}Cs and ^{137}Cs from parents' clothing or items removed from the workplace because exit monitors exist at nuclear power plants, and the extensive use of outerware that remains in the plant to prevent these types of incidents. It is also unlikely that children are exposed to ^{133}Cs from parents' clothing or items that have been removed from the workplace if the parents are employed in the mining, milling, or processing of pollucite ore. Other home exposures are unlikely since household items or products used in crafts, hobbies, or cottage industries do not contain significant amounts of cesium.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Few populations are likely to be exposed to high levels of ^{133}Cs. Persons residing in the vicinity of pollucite mines and workers employed in the mining, milling, and production of cesium may be exposed to higher levels than the general population.

Human exposure to ^{134}Cs and ^{137}Cs can be external, due to exposure from a radioactive cloud and contaminated environmental media after deposition, or internal, via inhalation and ingestion of contaminated food or drinking water. In both cases, populations residing near the source are exposed to potentially higher doses than populations far removed from the source. Humans residing near areas where nuclear weapons testing was previously conducted may have been exposed to higher doses of radiation from ^{134}Cs and ^{137}Cs, both internally and externally, than the general population. Populations residing in southern Utah and Nevada were exposed to radioactive cesium (and many other radionuclides) due to testing conducted at the Nevada Test Site (NTS). A total of 100 surface or near-surface tests with a total explosive yield of about 1 megaton were performed at the Nevada test site between 1951 and 1962. The dust from these tests also drifted over the continental United States, producing varying degrees of exposure for remote populations depending upon the meteorological conditions. For example, the greatest non-local fallout from one of the tests was received in New York state in 1953, some 2,000 miles away from the source, due to wet deposition (ATSDR 1999). About 500 underground tests were also performed at the NTS, but underground testing rarely leads to significant off-site contamination (ATSDR 1999). The EPA Office of Radiation and Indoor Air conducts off-site environmental monitoring around former U.S. nuclear test areas. The 1997 report concluded that the current exposure to populations

around the NTS from radionuclides, including ^{137}Cs and ^{134}Cs, was negligible in comparison to background levels (EPA 1999c).

Populations residing in the vicinity of nuclear power plants may also be exposed to higher levels of ^{134}Cs and ^{137}Cs than the general population due to airborne and liquid effluents from these plants. Persons employed in these facilities are also likely to be exposed to greater levels than the general population, particularly those employees who must handle radioactive waste material. However, despite the potential for exposure, increased body burdens of radioactive cesium have not been observed among the population of workers in nuclear facilities.

Human populations that received a large amount of fallout from the Chernobyl nuclear accident are also potentially exposed to high levels of radiocesium. These areas were primarily located in the Ukraine and northern Europe that received a great deal of rainfall in the weeks following the accident. Not including the 30-km exclusionary zone, an area of approximately $2.4x10^4$ km^2 near the plant was contaminated with ^{137}Cs at a deposition density $>5.4x10^{-5}$ Ci/m^2 (UNSCEAR 1996). Within the exclusionary zone, the contamination density may have been a factor of about 100 greater in limited areas (UNSCEAR 1996). The Bryansk-Belarus region, about 200 km northeast of the reactor, had deposition densities as high as $1.3x10^{-4}$ Ci/m^2 and the Kaluga-Tula-Orel location, approximately 500 km northeast of the reactor had deposition densities of about $1.6x10^{-5}$ Ci/m^2 (ATSDR 1999). While cesium is not considerably taken up by the roots of vascular plants, the deposition of radioactive debris on flora with large surface areas such as lichens or moss is significant (see Section 6.4.4). As a result, animals that feed on this vegetation such as caribou or reindeer may ingest large quantities of radiocesium. Nordic or Eskimo populations which use these animals as an important source for food are exposed to larger quantities of ^{137}Cs and ^{134}Cs than the general population (Allaye-Chan et al. 1990; WHO 1983). The average concentration of ^{137}Cs in the skeletal muscle of a herd of caribou from Alaska was in the range of 76–133 Bq/kg in 1987 (Allaye-Chan et al. 1990). According to the National Council on Radiation Protection and Measurements, the maximum nonoccupational radiocesium intake is 300,666 Bq/year ($8.1x10^6$ pCi/year) for adults (NCRP 1977). At the maximum average skeletal muscle concentration (133 Bq/kg), an annual consumption of over 2,260 kg would be required to reach this limit (assuming no other intake sources). Using the mean ^{137}Cs level of 900 Bq/kg in the muscle of reindeer obtained from northern Sweden in 1986–1987 (Ahman and Ahman 1994), over 330 kg of contaminated meat would have to be consumed to reach the maximum intake level. Populations residing in the Marshall Islands were exposed to higher levels of ^{137}Cs than the general population due to nuclear weapons testing conducted by the United States from 1946 to 1958. Using radiological surveys from 1978 and 1985–1986, the AVDI of ^{137}Cs due to food ingestion was

estimated for seven age groups residing in the Rongelap Atoll (Robinson and Phillips 1989). The AVDI in pCi/day were as follows: 0–3 months of age, 424; 4–8 months of age, 556; 9 months to 1.4 years of age 773; 1.5–3 years of age, 517; 4–11 years of age, 594; 12–17 years of age, 761; over 18 years of age, 1,085 (Robinson and Phillips 1989). This corresponds to an annual intake of 3.96×10^5 pCi/year, for adults over 18 years of age.

6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cesium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cesium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.8.1 Identification of Data Needs

Physical and Chemical Properties. As shown in Table 4-2, the relevant physical and chemical properties of cesium and its compounds are known (Burt 1993; Lide 1998). In general, cesium compounds are very water soluble and primarily exist in ionic form in aqueous environments. Cesium adsorbs strongly to soils and is not very mobile (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). The radioactive decay modes of the two most important cesium isotopes, ^{134}Cs and ^{137}Cs, are also well understood (ICRP 1983) and no physical or chemical data needs are required to permit the prediction of the environmental fate of cesium.

Production, Import/Export, Use, Release, and Disposal. Knowledge of a chemical's production volume is important because it may indicate possible environmental contamination and human exposure. If a chemical's production volume is high, there is an increased probability of general population exposure via consumer products and environmental sources such as air, drinking water, and food. Data concerning production volumes, import, and use of commercially significant cesium compounds are not available. No information was found for cesium in the TRI. According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. This database will be updated yearly and should provide a list of industrial production facilities and emissions. The United States is 100% import reliant for ^{133}Cs. There are no salient statistics such as production volume, consumption, or import/export volumes of cesium. Although there is no information regarding the countries shipping cesium or cesium compounds to the United States, it is believed that Canada is the major source of cesium (USGS 1999). Data regarding the U.S. production (if any) of cesium and its compounds as well as import/export statistics would be useful. The amount of ^{134}Cs and ^{137}Cs released in airborne and liquid effluents from nuclear power plants in the United States is known (NRC 1993b).

Environmental Fate. Information is available to permit assessment of the environmental fate and transport of cesium in air (Hirose et al. 1993; Rybacek et al. 1994), water (Burt 1993; WHO 1983), and sediment and soil (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). Cesium compounds are water soluble, and only one oxidation state (+1) is observed under environmental conditions (Burt 1993). Cesium released to the atmosphere may be carried long distances before being deposited onto soil and water surfaces by wet and dry deposition (Hirose et al. 1993). Most of the cesium released to water will adsorb to suspended solids in the water column and ultimately be deposited in the sediment core. In soil surfaces, cesium is strongly adsorbed in the upper layers and generally has very low mobility (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). This is particularly true for soils with a high potassium content or soils rich in clay. The radioactive decay modes of the two most important cesium isotopes, ^{134}Cs and ^{137}Cs, have been described in Chapter 4 (ICRP 1983).

Bioavailability from Environmental Media. The bioavailability of cesium in environmental media is well understood and no data needs are required at this time. For the most part, cesium is adsorbed strongly in the surface of most soils and is not readily bioavailable (Paasikallio 1999). In a study in which 102 healthy volunteers were fed ^{134}Cs-contaminated soil pellets, only about 1% of the original intake was absorbed, and on average, about 60% of the original amount was excreted within 48 hours

(LeRoy et al. 1966). In peaty or podzolic soils with a low clay content, cesium is reversibly adsorbed to the organic fraction and is expected to be in bioavailable form (Sanchez et al. 1999). Cesium uptake in vascular plants has been demonstrated (Djingova et al. 1995; Willey and Martin 1995). Increasing the clay or potassium content results in lower uptake by plants (Shenber and Johanson 1992). It has also been shown that fish residing in waters with high concentrations of humic material and potassium, such as oceans, have lower whole-body cesium concentrations than fish in freshwater where the concentration of dissolved potassium is lower given the same cesium concentration in the water (WHO 1983).

Food Chain Bioaccumulation. Cesium bioaccumulates in both aquatic and terrestrial food chains (Rowan et al. 1998; WHO 1983). Mean BCFs for ^{137}Cs of 146, 124, and 63 were reported for fish, brown macroalgae, and molluscs, respectively (Fisher et al. 1999). Cesium accumulation in aquatic organisms occurs from both food sources and cesium dissolved in the water column or adsorbed to suspended solids and sediments. The lichen-caribou-human food chain is an important route of human exposure and has been well studied (Allaye-Chan et al. 1990; WHO 1983). No additional data are required to assess the potential for human exposure to cesium through food chain bioaccumulation.

Exposure Levels in Environmental Media. Stable cesium and radioactive cesium have been detected in air (Ajdacic and Martic 1990; Chiavarini et al. 1994; Dasch and Wolff 1989; Rybacek et al. 1994; Todorovic et al. 1999), water (Asubiojo et al. 1997; DOE 1992; Fisher et al. 1999; Prister et al. 1990; Strezov et al. 1999; WHO 1983), soil/sediment (Burt 1993; DOE 1992, 1998a; Ruse and Peart 2000; WHO 1983), and food (Ahman et al. 1994; Bibak et al. 1998; Mondon and Walters 1990). Due to the large surface area of lichens and moss, they can collect a great deal of atmospheric nuclear fallout and often have high concentrations of ^{134}Cs and ^{137}Cs (Papastefanou et al. 1992; Penttila et al. 1993). Grazing animals such as reindeer and caribou that feed on large quantities of lichens or moss may potentially ingest large amounts of radioactive cesium and this may be transferred to humans who consume these animals as a meat source (Allaye-Chan et al. 1990; WHO 1983). Continued monitoring data on ^{134}Cs and ^{137}Cs in environmental media are needed to extend knowledge of human exposure to these radionuclides.

Exposure Levels in Humans. Monitoring data exist for the levels of ^{134}Cs and ^{137}Cs in human populations (Glowiak et al. 1977b; Lokobauer et al. 1988; Rabitsch et al. 1991; Rahola and Suomela 1998; Ropolo and Cesana 1997). Most of the current data is for areas of eastern Europe and Russia. Limited data are available regarding the levels of ^{133}Cs in humans (Hewitt 1988). Stable cesium was detected in the urine of U.S. residents at a geometric mean concentration of 4.2 µg/L (CDC 2001). More information regarding the background concentration of ^{133}Cs in human populations would be useful, but

given the ubiquitous distribution of cesium at low levels in the environment, background levels are unlikely to approach levels that would reflect cesium toxicity. Since the levels of radioactivity change over time, continued monitoring data on the levels of ^{134}Cs and ^{137}Cs in humans are needed in order to interpret health effects that may occur at current exposure levels.

Exposures of Children. Children, like adults, are not expected to be over exposed to ^{133}Cs. Existing data show that children are exposed to ^{134}Cs and ^{137}Cs after major releases. These exposures are primarily related to dietary intake and the intake of contaminated milk (Lokobauer et al. 1988). Dietary consumption patterns suggest that the weight-adjusted intake of radioactive cesium for children may be greater than for adults following a nuclear release (Lokobauer et al. 1988). In a study of females from northern Sweden, ^{137}Cs was detected in breast milk from 8 out of 12 mothers at concentrations of 7.3–178.4 pCi/kg (Johansson et al. 1998). The infants of these mothers who were being breast-fed had whole-body levels of ^{137}Cs in the range of 45.9–675.7 pCi/kg (Johansson et al. 1998). Additional data regarding the transfer of ^{134}Cs and ^{137}Cs from breast milk and contaminated pasteurized milk samples to children, as well as body burden studies on children, would be useful in assessing the potential risk that these radionuclides would pose following a major release.

Child health data needs relating to susceptibility are discussed in Section 3.12.2, Identification of Data Needs: Children's Susceptibility.

Exposure Registries. Cesium is currently one of the chemicals for which a subregistry has been established in the National Exposure Registry. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to cesium.

6.8.2 Ongoing Studies

The database of federal research programs in progress (FEDRIP 2000) indicates several current projects that may fill some existing data gaps and add to the current database of knowledge. Studies are in progress that seek to identify methods for more effective removal of radioactive cesium from contaminated soils and wastes. Phytotech Incorporated is conducting a study regarding the mobilization and removal of strontium and cesium from soil by chemical treatment and phytoremediation. Rufus L. Chaney from the Beltsville Agricultural Research Center in Beltsville, Maryland is researching the feasibility of developing plants, soil, and plant management practices that can cost-effectively

phytoextract radionuclides and heavy metals from contaminated soils. Dr. Heit from the U.S. DOE is conducting measurements regarding the fallout of nuclear debris from both atmospheric weapons tests and accidental atmospheric releases to determine the mechanisms of transport and deposition and to verify and correct fallout models. Work is being performed by Dr. Kinkead at the Los Alamos National Laboratory regarding the separation of cesium and strontium from high level radioactive waste. Dr. Leon Kochian from the Agricultural Research Center in Ithaca, New York is investigating the bioaccumulation of ^{137}Cs in plants grown in contaminated soils.

7. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, measuring, and/or monitoring cesium, its metabolites, and other biomarkers of exposure and effect to cesium. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits and/or to improve accuracy and precision.

7.1 BIOLOGICAL MATERIALS

Entry of cesium and its radioisotopes into the human body occurs by ingestion, inhalation, or penetration through skin (IAEA 1988; NCRP 1977, 1985). The amounts of cesium in the body can be assessed by bioassay *(in vivo* and/or *in vitro)* measurements. *In vivo* measurements are made with whole-body counters. *In vivo* measurement techniques are commonly used to measure body burdens of cesium radioisotopes, but can not be used to measure the stable isotope of cesium. *In vitro* measurements provide an indirect estimate of internally deposited cesium (both the stable and radioactive isotopes), utilizing techniques that measure cesium in body fluids, feces, or other human samples (Gautier 1983). Examples of these analytical techniques are given in NCRP Report No. 87 (1987) and are also listed in Table 7-1 for stable cesium and Table 7-2 for radioactive cesium.

7.1.1 *In Vivo* Cesium Measurements

In vivo measurement techniques are the most direct and widely used approach for assessing the burden of cesium radioisotopes within the body. The *in vivo* measurement of these radioisotopes within the body is performed with various radiation detectors and associated electronic devices that are collectively known as whole-body counters. These radiation detectors commonly utilize sodium iodide (NaI), hyperpure germanium, and organic liquid scintillation detectors to measure the 605 and 796 keV gamma rays from the decay of ^{134}Cs, and 662 keV gamma rays that are emitted from the decay of ^{137}Cs (Palmer et al. 1976). Because of the relatively low attenuation of the gamma rays emitted from ^{134}Cs and ^{137}Cs by most tissues

Table 7-1. Analytical Methods for Determining Cesium in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine	Sample dried, ashed and irradiated	INAA	0.00001 µg/g	No data	Oughton and Day 1993
Soft tissue	Sample ashed and then concentrated by precipitation with AMP, extracted with sodium phenylboron	Flame photometry	0.005 µg/g	96–99%	Feldman and Rains 1964
Feces	Sample dried, ashed, and irradiated	INAA	0.00001 µg/g	No data	Oughton and Day 1993

AMP = ammonium molybdophosphate; INAA = instrument neutron activated analysis

Table 7-2. Analytical Methods for Determining Radiocesium in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine	Sample is acidified and concentrated on a KCFC column	γ-spectrometry with NaI detector	6 pCi/L	98%	Boni 1966
Urine	Sample transferred to Marinelli beaker and counted	γ-spectrometry with NaI detector	100 pCi/L	98%	Gautier 1983
Urine	Sample transferred to Marinelli beaker and counted	γ-spectrometry with NaI(Tl) detector	2 pCi/L	No data	Cahill and Wheeler 1968
Soft tissue	Sample wet-ashed	γ-spectrometry	No data	No data	Baratta et al. 1969
Soft tissue	Sample directly counted in detector	γ-spectrometry	5 pCi/g	No data	Rabon and Johnson 1973
Soft tissue	Sample digested in acid, oxidized with $HClO_4$, concentrated by precipitated with AMP, purified by resin column, precipitated with hexachloroplatinic acid	β-counter	0.1 pCi/g	40–85%	Nevissi 1992
Feces	Sample directly counted in detector	γ-spectrometry with NaI detector	0.14 nCi/L	No data	Lipsztein et al. 1991
Human milk	Sample directly counted in detector	γ-spectrometry with NaI detector	0.001 pCi/L	No data	Risica et al. 1994

KCFC = potassium cobalt ferrocyanide

and the uniform distribution of cesium throughout the human body, cesium radioisotopes can easily be detected and quantitated using whole-body counting techniques (NCRP 1987; Palmer et al. 1976; Sun et al. 1997). Many configurations of the whole-body counter and scanning methods have been utilized, ranging from unshielded single-crystal field detectors to shielded, multi-detector scanning detectors (IAEA 1962, 1970, 1972, 1976, 1985; NCRP 1987; Palmer et al. 1976). Where appropriate, shielding of the room that houses the whole-body counter and/or the detector is often used to increase the detection sensitivity of the equipment by minimizing background radiation. Additionally, care must be exercised to insure that external contamination with radioactive cesium or other gamma-emitting radioisotopes are not present on the clothing or skin of the individual to be scanned. *In vitro* measurements of cesium (see Section 7.1.2) are used in conjunction with whole-body counting when monitoring individuals working with cesium, especially in conjunction with the assessment of individuals who have experienced accidental exposures to cesium (Bhat et al. 1973).

Calibration of whole-body counts is achieved through the use of tissue-equivalent phantoms. These phantoms are constructed to mimic the shape and density of the anatomical structure using tissue equivalent materials such as water-filled canisters or masonite (Barnaby and Smith 1971; Bhat et al. 1973; Sun et al. 1997). For example, the bottle mannequin absorber (BOMAB) consists of a series of water-filled polyethylene canisters constructed into seated or reclined human forms (Sun et al. 1997). Cesium standards are measured either as point sources along the phantom or dissolved within the water-filled canisters. Comparisons of the actual counts obtained from the phantom to the known activity of the cesium standards are used to determine the efficiency of the counting technique and, thus, provide the basis for calibrating the technique.

Assessment of short and long-term retention of cesium radioisotopes take into account the turnover rate for cesium within the human body. Although the physical half-life of ^{137}Cs is 30 years, the biological and effective half-life of cesium within the body is approximately 110 days (NCRP 1987; Rundo and Newton 1964). This relatively high turnover rate for cesium within the body is due to the high solubility of cesium in aqueous media that allows for the rapid uptake (e.g., absorption of ingested cesium through the gut) and elimination of cesium into and from the body (e.g., excreted through urine) (NCRP 1987). For acute and chronic exposures to cesium, the estimates of cesium retention are determined from direct, multiple whole-body measurements. Several retention models for cesium in the human body have been developed to aid in estimating the short- and long-term retention of cesium based on whole-body measurement techniques (ICRP 1979, 1989, 1993; NCRP 1987; Sun et al. 1997). However, direct comparisons of cesium body burdens and clearance rates among laboratories can be complicated by the

differing whole-body measurement techniques, calibration methods, and methods used to account for normal background radiation counts that are utilized within the different laboratories.

7.1.2 *In Vitro* Cesium Measurements

In vitro analyses of cesium are routinely performed in support of an *in vivo* monitoring program or in situations where direct *in vivo* measurements can not be obtained. Urinalysis is the preferred sample for *in vitro* analyses of cesium, although other sample types, such as feces, tissue, bone, or blood, can also be analyzed. Urinalysis is an optimum method for assessing the clearance of soluble cesium. Fecal analysis is used to assess the clearance of ingested, insoluble cesium (Baratta et al. 1969; Gautier 1983; Ide and McInroy 1975; NCRP 1987).

The *in vitro* analysis of the stable isotope of cesium, ^{133}Cs, in human samples (e.g., urine, tissue, feces) is performed by a number of methods that have the selectivity and sensitivity to measure cesium in biological matrices. These methods include spectrophotometry, instrumental neutron activation analysis (INAA), and inductively coupled plasma mass spectrometry (ICP-MS) (Dreizen et al. 1970; Iyengar and Woittiez 1988; Paschal et al. 1996). Of these methods, the INAA and ICP-MS methods offer the greatest detection sensitivity and are the preferred method of analysis for cesium in human samples (Iyengar and Woittiez 1988).

For the *in vitro* analysis of the cesium radioisotopes ^{134}Cs and ^{137}Cs in human samples, a number of analytical methods can be used to measure the cesium radioisotopes directly in the samples without requiring an extensive sample preparation procedure. In the radiochemical analysis of cesium in urine, a 24-hour urine collection (approximately 2 L) is obtained, followed by the transfer of a 1 L aliquot to a Marinelli beaker for counting in a gamma ray spectrometer (Gautier 1983). This simple procedure offers high recoveries of cesium (98%) and the minimum detection sensitivity (100 pCi/L) that is required to evaluate individuals for exposures to radioactive cesium (Gautier 1983). Similar methods are also used for the analysis of cesium radioisotopes in tissues, feces, and blood (Table 7-1). Mass spectrometry techniques have also been employed to measure cesium radioisotopes in human samples.

Accuracy of *in vivo* and *in vitro* measurements of cesium is determined through the use of standard, certified solutions or radioactive sources with known concentrations or activities of cesium. National Institute of Standards and Technology (NIST) traceable standards for ^{133}Cs can be obtained through a number of commercial sources. The primary source of certified cesium radioisotope standards is the

NIST. Gamma ray point sources for ^{137}Cs (standard reference material [SRM] 4200, 60,000 Bq [1.6 µCi] and SRM 4207, 300,000 Bq [56 µCi]) and standard solutions of ^{137}Cs (SRM 4233, 600,000 Bq/g [16 µCi/g]) are available from NIST.

7.2 ENVIRONMENTAL SAMPLES

Two common approaches are available for measuring cesium radioisotopes in the environment. Cesium radioisotopes can either be measured directly in the field (*in situ*) using portable survey instruments, or samples can be procured from the field and returned to the laboratory for quantitation of cesium. However, quantitation of the stable cesium isotope in environmental samples is generally conducted in the laboratory.

7.2.1 Field Measurements of Cesium

In situ measurement techniques are useful for the rapid characterization of radionuclide contamination in the environment, such as soils, sediments, and vegetation, and for monitoring personnel for exposure to radionuclides. The measurement of gamma ray-emitting radionuclides such as ^{134}Cs and ^{137}Cs in the environment is conducted with portable survey instruments such as Gieger-Mueller detectors, sodium iodide scintillation detectors, and gamma-ray spectrometers. The use of gamma-spectrometers in field survey equipment is preferred for measuring cesium in the field because of its energy selectivity and detection sensitivity. The relatively high energy and penetrance of the gamma-ray that is emitted during the decay of ^{134}Cs and ^{137}Cs provide an advantage for assessing the level of cesium. These gamma-ray spectrometers are equipped with a high purity germanium detector that is able to separate the 602, 662, and 796 keV gamma-rays emitted from ^{134}Cs and ^{137}Cs from the gamma-rays emitted from other radionuclides; for example, ^{40}K (NRC 1997). Minimum detectable activities (MDAs) of 0.005 Bq/g (0.05 pCi/g) for ^{137}Cs are routinely achieved using p-type germanium gamma spectrometers with 10 minute counting times (NRC 1997). Computational methods have been derived to aid in determining the concentrations and distributions of ^{134}Cs and ^{137}Cs in different soil types and depths (Fülöp and Ragan 1997; Hillmann et al. 1996; NRC 1997). The concentrations and distributions of ^{134}Cs and ^{137}Cs that have been derived from the computational analysis of the survey data are often verified by laboratory-based analyses of soil samples procured from the survey area.

7.2.2 Laboratory Analysis of Environmental Samples

Analytical methods for measuring cesium and cesium radioisotopes in environmental samples (e.g. air, water, soil, and biota) are summarized in Tables 7-3 (^{133}Cs) and 7-4 (^{134}Cs, ^{137}Cs). The methods that are commonly used in the analysis of ^{133}Cs are based on instrumental analytical techniques such as spectrophotometry, instrumental neutron activation analysis, and mass spectrometry. The analysis of ^{134}Cs and ^{137}Cs can be determined either as total mass or total activity, depending on the analytical technique that is used. Typically, radiochemical methods of analysis employing gamma spectrometry techniques are used to quantitate ^{134}Cs and ^{137}Cs in environmental samples. However, spectrophotometric and mass spectrometry techniques have been used to determine the total mass of ^{134}Cs and ^{137}Cs in samples. Using the specific activity of ^{137}Cs (89 μCi/μg), it can be deduced that a sample with activity of 1 μCi of ^{137}Cs contains roughly 0.011 μg of ^{137}Cs.

The analysis of cesium in air is based on the measurement of cesium in aerosols or particles that become trapped on cellulose or glass fiber filters after a measured amount of air is pulled through the filters. For the analysis for ^{133}Cs, the filter is solvent extracted and the extracted metals are analyzed by INAA (Gone et al. 2000). Analysis of ^{134}Cs and ^{137}Cs can be performed directly from the filter, or by following some sample preparation (e.g., ashing or solvent extraction), using gamma spectrometry (Kanapilly et al. 1983; Kolb 1971; Krieger et al. 1976).

For the analysis of cesium in water, a broad array of available sample preparation and detection methodologies (see Tables 7-3 and 7-4). Different standardized methods that can directly measure cesium or its radioactive isotopes within a water sample using INAA or radiochemical techniques with minimal sample preparation and good detection sensitivities (10–20 pCi/L), precision (4–9%), and bias (-5–1%) (ASTM 1999; EPA 1980). Other methods are available that preconcentrate cesium from natural or potable waters when interfering impurities are present or the activity of the cesium radioisotopes are too low (<30 pCi/L) for quantitation by gamma spectrometry (APHA 1998; Frigieri et al. 1980). This preconcentration of cesium can be achieved either through precipitation with molybdate compounds, for example, or through chromatographic techniques using columns packed with resins that specifically bind cesium (EPA 1980; Frigieri et al. 1980; Gaur 1996; Petrow and Levine 1967).

The quantity of cesium and its radioisotopes in soil, sediments, vegetation, and biota is determined using detection methods similar to those described above (Tables 7-3 and 7-4). Analysis of the stable cesium isotope by INAA and ICP-MS requires either digesting the sample in acid or ashing the sample before

Table 7-3. Analytical Methods for Determining Cesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Water	Sample is purified by passage through Dowex 50-X8 resin, concentrated with NCFC column	Electrothermal-AA	1 µg/L	99% at 10–100 µg/L	Frigieri et al. 1980
River water	Sample concentrated by precipitation with AMP, purified by extraction with sodium tetraphenylboron	Flame photometry	0.010 µg/L	94.5%	Feldman and Rains 1964
River water	Sample is purified by passage through Dowex 50-X8 resin, concentrated with NCFC column	Electrothermal-AA	1 µg/L	99% at 10–100 µg/L	Frigieri et al. 1980
Lake water	Filtered samples were irradiated	INAA	0.010 µg/L	90%	Hakonson and Whicker 1975
Sea water	Sample was precipitated with sodium tetraphenyl-borate, and the precipitate was neutron irradiated	INAA	0.008 µg/L	92%	Taskaev 1987
Sea water	Sample precipitated with AMP, purified by extraction with sodium tetraphenyl-boron	Flame photometry	0.010 µg/L	94.5%	Feldman and Rains 1964

Mineral and thermal waters	Direct aspiration of sample into graphite furnace	Graphite furnace-AA	0.00185 µg/L	92.3–100.9%	Bermejo-Barrera et al. 1989

Table 7-3. Analytical Methods for Determining Cesium in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Groundwater	Sample purified by ultracentrifugation	ICP-MS	0.010 µg/L	No data	Probst et al. 1995
Groundwater	Sample purified by ultracentrifugation	ICP-AES	0.010 µg/L	No data	Probst et al. 1995
Soil	Sample was pre-ashed, digested with acid	Electrothermal-AA	0.09 mg/g	80–85%	Anderson et al. 1996
Soil	Sample was dried, ground, and irradiated	INAA	0.003 ng/g	No data	Oughton and Day 1993
Soil	Sample digested with acid	ICP-MS	0.011 µg/g	95%	Robb et al. 1995
Sediment	Sample was dried and irradiated	INAA	0.010 µg/g	90%	Hakonson and Whicker 1975
Silicate rock	Sample digested in HF/H_2SO_4	Graphite furnace-AA	0.05 µg/L	76%	Grobenski et al. 1983
Vegetation	Sample ashed and irradiated	INAA	0.00001 µg/g	No data	Oughton and Day 1993
Vegetation	Sample prepared by microwave digestion	ICP-MS	0.00002 µg/g	95–105%	Dombovári et al. 2000

AA = atomic absorption; AMP = ammonium molybdophosphate; ICP-AES = inductively coupled plasma-atomic emission spectrometry; ICP-MS = inductively coupled plasma-mass spectrometry; INAA = instrumental neutron activation analysis; NCFC = ammonium hexacyanocobalt ferrate

Table 7-4. Analytical Methods for Determining Radiocesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air (occupational)	Sample filter was solvent extracted	Scintillation counter with NaI detector	No data	95%	Kanapilly et al. 1983
Air (ambient)	Sample filter digested in acid, cesium precipitated with chloroplatinate	γ-spectrometry with Ge/Li detector	0.01 fCi/m^3	80%	Krieger et al. 1976
Drinking water	Direct count of sample	γ-spectrometry with Ge/Li detector	10 pCi/L	no data	EPA 1980
Drinking water	Direct count of sample	γ-spectrometry with Ge detector	<2 pCi/L	92–100%	APHA 1998
Fresh water	Sample concentrated with Dowex 1x8/KCFC mixed ion exchange column	γ-spectrometry with NaI detector	3 pCi/L	99%	Boni 1966
River water	Sample precipitated with AMP, concentrated with Dowex-50 cation exchange column	Scintillation counter with NaI detector	<7 fCi/L	99%	Kahn et al. 1957
River water	Sample concentrated on Dowex 50W-X8 column	γ-spectrometry with Ge(Li) detector	2 pCi/L (50 L sample)	97%	Luetzelschwab 1976
Lake water	Sample concentrated on ACFC column	γ-spectrometry with NaI(Tl) detector	No data	97%	Eyman and Kevern 1975
Water and waste water	Direct count of sample	γ-spectrometry with Ge/Li detector	<2 pCi/L	92–100% at 2–94 pCi/L	ASTM 1999

Table 7-4. Analytical Methods for Determining Radiocesium in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Sea water	Sample purified by passage through chelating resin, concentrated with KCFC ion exchange column	γ-spectrometry with NaI detector	0.07 pCi/L	98%	Boni 1966
Soil	Sample dried and crushed	γ-spectrometry with Ge(Li) detector	0.05 pCi/g	No data	Arnalds et al. 1989
Soil	Sample mixed with 5% Ag and compressed into disc	GDMS	0.2 pg/g	No data	Betti et al. 1996
Sediment	Sample extracted with acid, concentrated by precipitation with AMP, solvent extracted with sodium tetraphenylboron	γ-spectrometry with Ge(Li) detector	No data	96%	Eyman and Kevern 1975
Biota	Sample ashed, oxidized with $HclO_4$, concentrated by precipitation with AMP, puriifed by resin column, precipitated with hexachloroplatinic acid	β-counter	0.1 pCi/g	No data	Nevissi 1992

ACFC = ammonium hexacyanocobalt ferrate; AMP=ammonium molybdophosphate; ICP-MS = inductively coupled plasma-mass spectrometry; KCFC = potassium cobalt ferrocyanide; GDMS = glow discharge mass spectrometry

analysis. In some cases where interfering compounds or materials may be present, additional sample concentration or purification may be required. For the radioisotopes of cesium, direct detection of ^{134}Cs and ^{137}Cs within the neat sample can be performed using gamma spectrometry detection methods.

The detection limits, accuracy, and precision of any analytical methodology are important parameters in determining the appropriateness of a method to quantitate a specific analyte at the desired level of sensitivity within a particular matrix. The MDA has been adopted to refer to the intrinsic detection capability of a measurement procedure (sampling through data reduction and reporting) to aid in determining which method is best suited for the required sample quantitation (NRC 1984). Several factors influence the MDA, including background count rates, size or concentration of sample, detector sensitivity, recovery of desired analyte during sample isolation and purification, level of interfering contaminants, and particularly, counting time. Because of these variables, the MDAs will vary between laboratories utilizing the same or similar measurement procedures.

The accuracy of a measurement technique in determining the quantity of a particular analyte in environmental samples is greatly dependent on the reliability of the calibrating technique. Thus, the availability of standard, certified radiation sources with known concentrations of cesium and its radioisotopes is required in order to insure the reliability of the calibration methods and accuracy of cesium measurements in environmental samples. NIST traceable standards for ^{133}Cs can be obtained through a number of commercial sources. The primary source of certified cesium radioisotope standards is the NIST. Gamma ray point sources for ^{137}Cs (SRM 4200, 60,000 Bq [1.6 μCi] and SRM 4207, 300,000 Bq [56 μCi]) and standard solutions of ^{137}Cs (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST. SRMs are also available containing the stable (SRM 1944 [sediment], SRM 2710 and 2711 [soil]) and radioactive isotopes of cesium (SRM 4350 [sediment] and SRM 4357 [sediment]).

7.3 ADEQUACY OF THE DATABASE

Section 104(1)(5) of CERCLA directs the Administrator of ATSDR (in conjunction with the Administrator of EPA and agencies and programs of the Public Health Service) to access whether adequate information of the health effects of cesium is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cesium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

7.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Analytical methods with satisfactory sensitivity and precision are available to determine the levels of cesium and its radioisotopes in human tissues and body fluids.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Analytical methods with the required sensitivity and accuracy are available for measuring cesium, both total and isotopic, in environmental matrices (Tables 7-2 and 7-4). Knowledge of the levels of cesium in various environmental media, along with appropriate modeling (see Chapters 4 and 6), can be used to evaluate potential human exposures through inhalation and ingestion pathways.

Whether in the environment or in the human body, cesium radioisotopes will undergo radioactive decay to non-radioactive isotopes (see Chapter 4). Current analytical methods, such as mass spectrometry, have the necessary resolution and sensitivity to detect and quantitate these decay products.

7.3.2 Ongoing Studies

Current research trends in the quantitation of cesium and its radioisotopes are focused on improving the selectivity and detection sensitivity of cesium in biological and environmental samples. Mass spectrometry approaches, such as double focusing sector field inductively coupled mass spectrometry or time-of-flight selected ion monitoring systems, are being developed further to provide the required selectivity and sensitivity to rapidly measure cesium in the presence of other trace metals in complex environmental samples. Cesium-selective electrodes are being developed into a highly-selective, rapid detection technique for measuring cesium in environmental samples and waste streams. Current efforts are focused on the development of electrode membranes that contain cesium binding agents, such as crown ether derivatives. New cesium-selective resins, for example lanthanum-based resins or montmorillonite clays, are being developed and tested for selectivity and recovery of cesium.

The database of federal research programs in progress (FEDRIP) indicates several current projects that may fill some existing data gaps and add to the current database of knowledge. W.H. Aberth from Antek Incorporated, located in Palo Alto, California is attempting to increase the sensitivity to which cesium can be analyzed in human tissue through the use of a cluster ion gun in liquid dynamic secondary ion mass spectrometry (SIMS) (FEDRIP 2000).

8. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding stable cesium in air, water, and other media are summarized in Table 8-1. The regulations regarding radioactive cesium are summarized in Table 8-2.

No MRLs were derived for inhalation or oral exposure to stable or radioactive cesium. Two MRLs, derived by ATSDR (1999) for external exposure to ionizing radiation, are applicable to external exposure to radioisotopes of cesium. An MRL of 400 mrem (4.0 mSv) was derived for acute-duration external exposure (14 days or less), based on cognitive learning deficit in children who had been exposed to ionizing radiation at critical stages of fetal development (gestation weeks 8–15) during the atomic bombing of Hiroshima and Nagasaki (Schull et al. 1988). An MRL of 100 mrem/year (1.0 mSv/year) above background was derived for chronic-duration external exposure (365 days or more), based on the BEIR V (1990) report that the average annual effective ionizing radiation dose to the U.S. population is 360 mrem/year (3.6 mSv/year), a dose not expected to produce adverse health effects.

The EPA has not classified cesium for human carcinogenicity, nor has the EPA derived reference concentrations (RfCs) or reference doses (RfDs) for stable or radioactive cesium (IRIS 2000).

Table 8-1. Regulations and Guidelines Applicable to Cesium

Agency	Description	Information	Reference
INTERNATIONAL Guidelines:			
IARC		ND	IARC 2000
NATIONAL Regulations and Guidelines:			
a. Air:			
ACGIH	TWA–Cesium hydroxide based on upper respiratory tract and eye irritation	2 mg/m^3	ACGIH 2000
NIOSH	REL: TWA–Cesium hydroxide based on eye irritation	2 mg/m^3	NIOSH 2000
OSHA		ND	
EPA		ND	
b. Water		ND	
c. Food		ND	
d. Other		ND	
STATE		ND	

ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; IARC = International Agency for Research on Cancer; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; TWA = time weighted average

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cesium

Agency	Description	Information	Reference
INTERNATIONAL Guidelines:			
IARC		ND	IARC 2000
NATIONAL Regulations and Guidelines:			
a. Air:			
ACGIH		ND	ACGIH 2000
EPA	Detection limits for man-made beta particle and photon emitters	10 pCi/L	EPA 1999a 40 CFR 141.25
NIOSH		ND	NIOSH 2000
NRC	Effluent concentrations—air		NRC 1999
	Cesium 134	$2x10^{-10}$ µCi/mL	10 CFR 20 App B
	Cesium 137	$2x10^{-10}$ µCi/mL	
b. Water			
NRC	Effluent concentrations—water		NRC 1999
	Cesium 134	$9x10^{-7}$ µCi/ml	10 CFR 20 App B
	Cesium 137	$1x10^{-6}$ µCi/ml	
c. Food		ND	
d. other			
EPA	Concentration levels for environmental compliance—		EPA 1999 40 CFR 61 App E
	Cesium 134	$2.7x10^{-14}$ Ci/m^3	
	Cesium 137	$1.9x10^{-14}$ Ci/m^3	
	Carcinogenicity—slope factors[a]		EPA 1997b
	Lifetime risk per pCi—ingestion		
	Cesium 131	$1.80x10^{-13}$	
	Cesium 134	$4.73x10^{-11}$	
	Cesium 134 meta stable	$4.54x10^{-14}$	
	Cesium 135	$4.53x10^{-12}$	
	Cesium 136	$7.74x10^{-12}$	
	Cesium 137	$3.16x10^{-11}$	
	Cesium 137 plus disintegration products	$3.16x10^{-11}$	
	Cesium 138	$1.76x10^{-13}$	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cesium *(continued)*

Agency	Description	Information	Reference
NATIONAL (*cont.*)			
EPA (*cont.*)	Lifetime risk per pCi—inhalation		EPA 1997b
	Cesium 131	1.06×10^{-13}	
	Cesium 134	2.89×10^{-11}	
	Cesium 134 meta stable	3.10×10^{-14}	
	Cesium 135	2.71×10^{-12}	
	Cesium 136	4.65×10^{-12}	
	Cesium 137	1.91×10^{-11}	
	Cesium 137 plus disintegration products	1.91×10^{-11}	
	Cesium 138	1.30×10^{-13}	
	External exposure—risk/year per pCi/g soil		
	Cesium 131	2.34×10^{-9}	
	Cesium 134	5.88×10^{-6}	
	Cesium 134 meta stable	1.96×10^{-8}	
	Cesium 135	0	
	Cesium 136	8.13×10^{-6}	
	Cesium 137	0	
	Cesium 137 plus disintegration products	2.09×10^{-6}	
	Cesium 138	9.45×10^{-6}	
NRC	Occupational inhalation exposure		NRC 1999 10 CFR 20 App B
		100 µCi	
	ALIs	200 µCi	
	Cesium 134		
	Cesium 137	4×10^{-8} µCi/mL	
		6×10^{-8} µCi/mL	
	DACs		
	Cesium 134		
	Cesium 137		
	Quantities of licensed material requiring labeling—		NRC 1999 10 CFR App C
	Cesium 134	10 µCi	
	Cesium 137	10 µCi	
STATE			
A. Air			
Michigan	Gross beta particle activity—Cesium 134	15 pCi/L	MI Dept Environ Quality 2000
b. Water		ND	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cesium *(continued)*

Agency	Description	Information	Reference
STATE *(cont.)*			
c. Other			
Louisiana	Quantity required for consideration of need for emergency plan for responding to a release:		LA Dept Environ Quality 2000
	Cesium 134		
	Release fraction	0.01	
	Quantity	2,000 Ci	
	Cesium 137		
	Release fraction	0.01	
	Quantity	3,000 Ci	

[a]Radionuclide slope factors are calculated by EPA's Office of Radiation and Indoor Air (ORIA) to assist HEAST users with risk-related evaluations and decision-making at various stages of the remediation process. Ingestion and inhalation slope factors are central estimates in a linear model of the age-averaged, lifetime attributable radiation cancer incidence (fatal and nonfatal cancer) risk per unit of activity inhaled or ingested, expressed as risk/picocurie (pCi). External exposure slope factors are central estimates of the lifetime attributable radiation cancer incidence risk for each year of exposure to external radiation from photon-emitting radionuclides distributed uniformly in a thick layer of soil, and are expressed as risk/year per pCi/gram of soil.

ACGIH = American Conference of Governmental Industrial Hygienists; ALI = annual limitations on intake; CFR = Code of Federal Regulations; DAC = derived air concentrations; EPA = Environmental Protection Agency; IARC = International Agency for Research on Cancer; NIOSH = National Institute for Occupational Safety and Health; NRC = Nuclear Regulatory Commission; OSHA = Occupational Safety and Health Administration

9. REFERENCES

Aage HK, Korsbech U, Bargholz K, et al. 1999. A new technique for processing airborne gamma ray spectrometry data for mapping low level contaminations. Appl Radiat Isot 51:651-662.

Aarkrog A. 1994. Doses from the Chernobyl accident to the nordic populations. In: Dahlgaard H, ed. Studies in environmental science. Nordic radioecology: The transfer of radionuclides through nordic ecosystems to man. New York, NY: Elsevier Science Publishers. Vol 62 433-456.

Aarkrog A, Baxter MS, Bettencourt AO, et al. 1997. A comparison of doses from ^{137}Cs and ^{210}Po in marine food: A major international study. J Environ Radioact 34(1):69-90.

Abdul-Majid S, Abulfaraj W. 1992. Radioactivity concentration in soil in Jeddah area, Saudi Arabia. J Environ Sci Health Part A 27(1):105-116.

*Abdul-Majid S, Abdul-Fattah A, Abulfaraj WH. 1992. Radiation dose assessment from ingestion pathway in Saudi Arabia. J Environ Sci Health A27(4):1045-1057.

*ACGIH. 2000. Threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

*Adinolfi M. 1985. The development of the human blood-CSF-brain barrier. Dev Med Child Neurol 27:532-537.

*Adlercreutz H. 1995. Phytoestrogens: Epidemiology and a possible role in cancer protection. Environ Health Perspect Suppl 103(7):103-112.

*Ahmad S, Waheed S, Mannan A, et al. 1994. Evaluation of trace elements in wheat and wheat by-products. J AOAC Int 77(1):11-18.

Ahman B. 1994. Body burden and distribution of ^{137}Cs in reindeer. Rangifer 14(1):23-28.

*Ahman B, Ahman G. 1994. Radiocesium in Swedish reindeer after the Chernobyl fallout: Seasonal variations and long-term decline. Health Phys 66(5):503-512.

*Ajdacic N, Martic M. 1990. Radionuclides in the ground layer of air. J Radioanal Nucl Chem, Art 140(2):295-300.

Albinsson Y, Engkvist I. 1989. Diffusion of americium, plutonium, uranium, neptunium, cesium, iodine and technetium in compacted sand-bentonite mixture. Goeteborg, Sweden: Chalmers University of Technology, Department of Nuclear Chemistry. SKB Tech. Rep. 89-22.

Al`Ferovich AA, Gotlib VYA, Pelevina II. 1996. Appearance of micronuclei in generation of hela cells irradiated at low doses. Izv Akad Nauk SSSR Ser Biol 3:261-264.

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*Cited in text

9. REFERENCES

*Allaye-Chan AC, White RG, Holleman DF, et al. 1990. Seasonal concentrations of cesium137 in rumen content, skeletal muscles and feces of caribou from the porcupine herd: lichen ingestion rates and implications for human consumption. Rangifer 3:17-24.

Allott RW, Kelly M, Hewitt CN. 1992. Behavior of urban dust contaminated by Chernobyl fallout: environmental half-lives and transfer coefficients. Environ Sci Technol 26:2142-2147.

Al-Sabati K. 1992. Monitoring the genotoxicity of radiocontaminants in Swedish lakes by fish micronuclei. Cytobios 70:101-106.

*Altman PL, Dittmer DS. 1974. Biological handbooks: Biology data book. Vol III. 2nd ed. Bethesda, MD: Federation of American Societies for Experimental Biology, 1987-2008, 2041.

Ames M, Gullu G, Olmez I. 1998. Atmospheric mercury in the vapor phase, and in fine and course particulate matter at Perch River, New York. Atmos Environ 32(5):865-872.

Amiro BD, Sheppard SC, Johnston FL, et al. 1996. Burning radionuclide question: What happens to iodine, cesium and chlorine in biomass fires? Sci Total Environ 187:93-103.

*Andersen ME, Krishnan K. 1994. Relating in vitro to in vivo exposures with physiologically based tissue dosimetry and tissue response models. In: Salem H, ed. Animal test alternatives: Refinement, reduction, replacement. New York: Marcel Dekker, Inc. 9-25.

*Andersen ME, Clewell HJ III, Gargas ML, et al. 1987. Physiologically based pharmacokinetics and the risk assessment process for methylene chloride. Toxicol Appl Pharmacol 87:185-205.

*Anderson P, Davidson CM, Littlejohn D, et al. 1996. The determination of caesium and silver in soil and fungla fruiting bodies by electrothermal atomic absorption spectrometry. Anal Chim Acta 327:53-60.

Anderson SH, Dodson GJ, Van Hook RI. 1976. Comparative retention of ^{60}Co, ^{109}Cd and ^{137}Cs following acute and chronic feeding in bobwhite quail. In: Radioecology and energy resources. Oakridge, TN: Environmental Science Division. Oak Ridge National Laboratory. Vol 1, 321-324.

Andersson I, Hansson I. 1989. Distribution of ^{137}Cs to different muscles and internal organs of lambs fed contaminated hay. Swed J Agric Res 19:93-98.

Andersson I, Hakansson J, Anner K. 1990. Transfer of ^{137}Cs from grain to muscle and internal organs of growing finishing pigs, and the effect of feeding bentonite. Swed J Agric Res 20:43-48.

Anisimov VN, Miretskii GI, Morozov VG, et al. 1992. The effect of synthetic immunomodulator thymogen on radiation carcinogenesis in rats. Vopr Onkol 38:481-488.

*APHA. 1998. 7120 Gamma-emitting radionuclides. Standard methods for the examination of water and wastewater, 20th ed. Washington, DC: American Public Health Association.

Arnalds O, Cutshall NH, Nielsen GA. 1989. Cesium137 in Montana soils. Health Phys 57:955-958.

*Arslan NC, Geard CR, Hall EJ. 1986. Low dose-rate effects of cesium-137 and iodine-125 on cell survival, cell progression, and chromosomal alterations. Am J Clin Oncol 9(2):114-115.

9. REFERENCES

Ashwood TL, Olsen CR. 1988. Pearl Harbor bombing attack: A contamination legacy revealed in the sedimentary record. Mar Pollut Bull 19(2):68-71.

Askbrant S, Melin J, Sandalls J, et al. 1996. Mobility of radionuclides in undisturbed and cultivated soils in Ukraine, Belarus and Russia six years after the Chernobyl fallout. J Environ Radioact 31(3):287-312.

Assimakopoulos PA, Ioannides KG, Pakou AA, et al. 1993. Transport of radiocaesium from a sheep's diet to its tissues. Sci Total Environ 136:1-11.

Astakhova LN, Anspaugh LR, Beebe GW, et al. 1998. Chernobyl-related thyroid cancer in children of Belarus: A case-control study. Radiat Res 150:349-356.

Astakhova LN, Demidchik EP, Kasakov VS, et al. 1993. Thyroid cancer in children of the Belarus Republic after the Chernobyl accident: Publisher's summary. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 425-427.

*ASTM. 1999. Annual book of ASTM standards, vol 11.02. Philadelphia, PA: American Society for Testing of Materials. ASTM, 290-300.

*Asubiojo OI, Nkono NA, Ogunsua AO, et al. 1997. Trace elements in drinking and groundwater samples in southern Nigeria. Sci Total Environ 208:1-8.

*ATSDR. 1989. Decision guide for identifying substance-specific data needs related to toxicological profiles: Notice. Agency for Toxic Substances and Disease Registry. Federal Register 54(174):37618-37634.

*ATSDR. 1990. Biomarkers of organ damage or dysfunction for the renal, hepatobiliary, and immune systems. Atlanta, GA: Subcommittee on Biomarkers of Organ Damage and Dysfunction. Agency for Toxic Substances and Disease Registry.

*ATSDR. 1999. Toxicological profile for ionizing radiation. Atlanta, GA: Agency for Toxic Substances and Disease Registry.

Baeza A, del Rio M, Miro C, et al. 1999. Recent evolution of the multi-isotopic radioactive content in ice of Livingston Island, Antarctica. Bull Environ Contam Toxicol 63:139-149.

Baker CE, Dunaway PB. 1969. Retention of ^{134}Cs as an index to metabolism in the cotton rat (Sigmodon hispidus). Health Phys 16:227-230.

*Balasem AN, Ali ASK. 1991. Establishment of dose-response relationships between doses of Cs-137 γ-rays and frequencies of micronuclei in human peripheral blood lymphocytes. Mutat Res 259:133-138.

Balduini W, Costa LG, Murphy SD. 1990. Potassium ions potentiate the muscarinic receptor-stimulated phosphoinositide metabolism in cerebral cortex slices: A comparison of neonatal and adult rats. Neurochem Res 15(1):33-39.

*Balonov MI. 1993. Overview of doses to the Soviet population from the Chernobyl accident and the protective actions applied. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 23-45.

9. REFERENCES

Balonov MI, Travnikova IG. 1993. Importance of diet and protective actions on internal dose from Cs radionuclides in inhabitants of the Chernobyl region. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 127-166.

*Baratta EJ, Apidianakis JC, Ferri ES. 1969. Cesium-137, lead-210 and polonium-210 concentrations in selected human tissues in the United States. Am Ind Hyg Assoc J 30:443-448.

Bard D, Verger P, Hubert P. 1997. Chernobyl, 10 years after: Health consequences. Epidemiol Rev 19(2):187-204.

Barnaby CF, Smith T. 1971. Calibration of a whole-body counter suitable for use in routine clinical investigations. Phys Med Biol 16:97-104.

*Barnes DG, Dourson M. 1988. Reference dose (RfD): Description and use in health risk assessments. Regul Toxicol Pharmacol 8:471-486.

*Barrie LA, Gregor D, Hargrave B, et al. 1992. Arctic contaminants: sources, occurrence and pathways. Sci Total Environ 122:1-74.

Barry SF. 1988. Epidemiology in occupational radiation exposure. Radiat Prot Dosim 25:85-92.

*Bartstra RW, Bentvelzen PAJ, Zoetelief J, et al. 1998. Induction of mammary tumors in rats by single-dose gamma irradiation at different ages. Radiat Res 150:442-450.

Baryakhtar V, Poyarkov V, Kholosha V, et al. 2000. The accident. In: Vargo GJ, ed. The Chernobyl accident. Columbus, OH: Batelle Press.

*Beaugelin-Seiller K, Baudin JP, Brottet D. 1994. Use of aquatic mosses for monitoring artificial radionuclides downstream of the nuclear power plant of Bugey (River Rhone, France). J Environ Radioact 24:217-233.

*Beck HL, Krey PW. 1983. Radiation exposures in Utah from Nevada nuclear tests. Science 220:18-24.

*Becker PR, Mackey EA, Demiralp R, et al. 1997. Concentrations of chlorinated hydrocarbons and trace elements in marine mammal tissues archived in the U.S. National Biomonitoring Specimen Bank. Chemosphere 34(9/10):2067-2098.

Begovic J, Stankovic S, Mitrovic R. 1980. Dynamics of Cs-137 distribution in the muscle tissue of swine by single and repeated contamination. In: Radiation protection: A systematic approach to safety: Proceedings of the 5th congress of the International Radiation Protection Society, Jerusalem, March 1980. New York, NY: Pergamon Press. Vol 2, 1029-1142.

Behne D, Gebner H. 1987. Effects of dietary K in the absorption and excretion of radiocesium in the rat. Health Phys 53(3):331-332.

*BEIR V. 1990. Health effects of exposure to low levels of ionizing radiation. Biological Effects of Ionizing Radiations. Washington, DC: National Academy Press.

*Bengtsson LG, Haversten Y, Svvensson KG. 1964. Maternal and infantile metabolism of cesium, In: Assessment of radioactivity in man. Vol II, Vienna: International Atomic Energy Agency, 21-32.

9. REFERENCES

Benjamin SA, Boecker BB, Chiffelle TL, et al. 1973. Neoplasia in beagle dogs after inhalation of $^{144}CeCl_3$. In: United States atomic energy commission symposium series. Springfield, VA: U.S. Atomic Energy Commision. Vol 29. 181-200.

Benjamin SA, Boecker BB, Cuddihy RG, et al. 1979. Nasal carcinomas in beagles after inhalation of relatively soluble forms of beta-emitting radionuclides. J Natl Cancer Inst 63(1):133-139.

Benjamin SA, Hahn FF, Chiffelle TL, et al. 1975. Occurrence of hemangiosarcomas in beagles with internally deposited radionuclides. Cancer Res 35:1745-1755.

Benoit G, Rozan TF, Patton PC, et al. 1999. Trace metals and radionuclides reveal sediment sources ans accumulation rates in Jordan Cove, Connecticut. Estuaries 22(1):65-80.

Berg D, Kollmer WE, Kriegel H, et al. 1987. Radioactive iodine and cesium in Bavarian citizens after the nuclear reactor accident in Chernobyl. Trace Subst Environ Health 21:219-225.

*Berger GS. 1994. Epidemiology of endometriosis. In: Berger GS, ed. Endometriosis: Advanced management and surgical techniques. New York, NY: Springer-Verlag.

Berger ME, Hurtado R, Dunlap J, et al. 1997. Accidental radiation injury to the hand: Anatomical and physiological considerations. Health Phys 72(3):343-348.

*Bermejo-Barrera P, Beceiro-Gonzalez E, Bermejo-Barrera A, et al. 1989. Determination of cesium in mineral and thermal waters by electrothermal atomic absorption spectrophotometry. Microchem J 40:103-108.

*Betti M, Giannarelli S, Hiernaut T, et al. 1996. Detection of trace radioisotopes in soil, sediment and vegetation by glow discharge mass spectrometry. Fresenius J Anal Chem 355:642-646.

Bettinelli M, Baffi C, Beone GM, et al. 2000. Soils and sediments analysis by spectroscopic techniques part II: Determination of trace elements by ICP-MS. Atom Spectrosc 21(2):60-70.

Beukema AA, Hekstra GP, Venema C. 1986. The Netherlands' environmental policy for the North Sea and Wadden Sea. Environ Monit Assess 7:117-155.

*Bhat IS, Hedge AG, Chandramouli S, et al. 1973. Evaluation of internal exposure to radionuclides of I, Cs, and Co, during maintenance operations on primary steam leak in a nuclear power station. Health Phys 25:135-139.

*Bibak A, Behrens A, Sturup S, et al. 1998. Concentrations of 63 major and trace elements in Danish agricultural crops measured by inductively coupled plasma mass spectrometry. 1. Onion (Allium cepa Hysam). J Agric Food Chem 46:3139-3145.

*Biedermann KA, Sun J, Giaccia AJ, et al. 1991. Acid mutation in mice confers hypersensitivity to ionizing radiation and deficiency DNA double-strand break repair. Proc Natl Acad Sci USA 88:1394-1397.

Bird GA, Hesslein RH, Mills KH, et al. 1998. Bioaccumulation of radionuclides in fertilized Canadian Shield Lake basins. Sci Total Environ 218:67-83.

*Boecker BB. 1969a. Comparison of ^{137}Cs metabolism in the beagle dog following inhalation and intravenous injection. Health Phys 16(6):785-788.

*Boecker BB. 1969b. The metabolism of ^{137}Cs inhaled as ^{137}CsCl by the beagle dog. Proc Soc Exp Biol Med 130(3):966-971.

Boecker BB. 1972. Toxicity of ^{137}CsCl in the beagle: Metabolism and dosimetry. Radiat Res 50:556-573.

Boecker BB, McClellan RO, Scott JK. 1969a. Retention and distribution of ^{137}Cs in the beagle dog following inhalation of ^{137}Cs in fused montmorillonite clay particles. In: Fission Product Inhalation Program, ed. Fission product inhalation program annual report 1968-69. Albuquerque, NM: Lovelace Foundation for Medical Research and Education, 105-110.

Boecker BB, Redman HC, Chiffelle TL, et al. 1969b. Toxicity of inhaled ^{137}CsCl in the beagle dog. II. In: Fission Product Inhalation Program, ed. Fission product inhalation program annual report 1968-69. Albuquerque, NM: Lovelace Foundation for Medical Research and Education, 36-54.

*Boecker BB, Thomas RG, McClellan RO. 1974. Retention patterns in beagle dogs repeatedly exposed to aerosols of ^{137}Cs-labeled fused clay particles. II. In: Inhalation Toxicology Research Institute, ed. Annual report of the Inhalation Toxicology Research Institute. Albuquerque, NM: Inhalation Toxicology Research Institute, 318-321.

*Boecker BB, Thomas RG, McClellan RO. 1977. Accumulation and retention of ^{137}Cs-labelled fused aluminosilicate particles by beagle dogs after repeated inhalation exposures. In: Inhaled particles IV: proceedings of an international symposium. New York, NY: Pergamon Press. Vol 1, 221-237.

*Bonatti V, Rolli A, Botti G. 1983. Recording of nonphasic action potentials of the right ventricle in long QT syndromes complicated by severe ventricular arrhythmias. Eur Heart J 4:168-179.

*Boni AL. 1966. Rapid ion exchange analysis of radiocesium in milk, urine, sea water, and environmental samples. Anal Chem 38:89-92.

Boni AL. 1969a. Organ specific inhibitor of mitosis in the amphibian kidney. Nature 222:1189.

*Boni AL. 1969b. Variations in the retention and excretion of ^{137}Cs with age and sex. Nature 222:1188-1189.

Borovec J. 1995. Changes in incidence of carcinoma in situ after the Chernobyl disaster in central Europe. Arch Environ Contam Toxicol 29:266-269.

Borzilov VA, Klepikova NV. 1993. Effect of meteorological conditions and release composition on radionuclide deposition after the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 47-68.

*Bose R, Pinsky C. 1981. Cesium impairs conditioned avoidance response (CAR) in mice and rats. Proc Can Fed Biol Soc 24:101.

9. REFERENCES

Bose R, Pinsky C. 1983a. Antipsychotic effects of cesium are suggested by mutual synergism between cesium chloride, chlorpromazine and haloperidol on conditioned avoidance response in mice. Res Commun Psychol Psychiatr Behav 8(4):317-329.

*Bose R, Pinsky C. 1983b. Cesium attenuates conditioned avoidance response in rats and mice. Pharmacol Biochem Behav 18:867-871.

*Bose R, Pinsky C. 1984. Central depressant action of cesium in mice. Psychopharmacology 84:80-84.

*Bose R, Pinsky C, Jasper SC, et al. 1981. Cesium on acquisition of conditioned avoidance response (CAR) and motor behavior in mice. Pharmacologist 23:151.

*Brachmann J, Scherlag BJ, Rosenshtraukh LV, et al. 1983. Bradycardia-dependent triggered activity: relevance to drug-induced multiform ventricular tachycardia. Circulation 68(4):846-856.

Bramham J, Riddell FG. 1994. Cesium uptake studies on human erythrocytes. J Inorg Biochem 53:169-176.

*Brandão-Mello CE, Oliveira AR, Valverde NJ, et al. 1991. Clinical and hematological aspects of 137Cs: The Goiania radiation accident. Health Phys 60(1):31-39.

Bretfeld G, Muth H, Oberhausen E. 1968. Organspezifische Caesium 137-aufnahme bei Ratten. Biophysik 4:343-355.

Brewer AK. 1984. The high pH therapy for cancer tests on mice and humans. Pharmacol Biochem Behav 21(Suppl. 1):1-5.

Brogger A, Reitan JB, Strand P, et al. 1996. Chromosome analysis of peripheral lymphocytes from persons exposed to radioactive fallout in Norway form the Chernobyl accident. Mutat Res 361:73-79.

*Brown JE, McDonald P, Parker A, et al. 1999. The vertical distribution of radionuclides in a Ribble Estuary saltmarsh: transport and deposition of radionuclides. J Environ Radioact 43:259-275.

Browning E. 1969. Toxicity of industrial metals. 2nd ed., London, Butterworths.

Brunn H, Georgii S, Eskens U. 1993. ^{137}Cesium and ^{134}cesium in roe deer from north and middle Hesse (Germany) subsequent to the reactor accident in Chernobyl. Bull Environ Contam Toxicol 51:633-639.

Brynildsen LI, Selnaes TD, Strand P, et al. 1996. Countermeasures for radiocesium in animal products in Norway after the Chernobyl accident - techniques, effectiveness, and costs. Health Phys 70(5):665-672.

*Bunzl K, Schimmack W. 1989. Associations between the fluctuations of the distribution coefficients of Cs, Zn, Sr, Co, Cd, Ce, Ru, Tc and I in the upper two horizons of a podzol forest soil. Chemosphere 18:2109-2120.

Burrows BA, Chalmers TC. 1990. Cesium-137/potassium-40 ratios in firewood ashes as a reflection of worldwide radioactive contamination of the environment. Ann N Y Acad Sci 609:334-339.

Burruel VR, Raabe OG, Wiley LM. 1997. In vitro fertilization rate of mouse oocytes with spermatozoa from the F1 offspring of males irradiated with 1.0 Gy ^{137}Cs γ-rays. Mutat Res 381:59-66.

9. REFERENCES

*Burt C. 1966. The genetic determination of differences in intelligence: A study of monozygotic twins reared together and apart. Br J Psychol 57(1+2):137-153.

*Burt RO. 1993. Cesium and cesium compounds. In: Kirk-Othmer encyclopedia of chemical technology. 4th ed. Vol 5. New York: John Wiley & Sons, 749-764.

*Buzulukov YP, Dobrynin YL. 1993. Release of radionuclides during the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 3-21.

*Cahill DF, Wheeler JK. 1968. The biological half-life of cesium-137 in children determined by urinary assay. Health Phys 14:293-297.

*Capar SG, Cunningham WC. 2000. Element and radionuclide concentrations in food: FDA total diet study 1991-1996. J AOAC Int 83(1):157-177.

Capra E, Drigo A, Menin A. 1989. Cesium-137 urinary excretion by northeastern (pordenone) Italian people following the Chernobyl nuclear accident. Health Phys 57(1):99-106.

Castronovo FP. 1999. Teratogen update: Radiation and Chernobyl. Teratology 60:100-106.

Caywood K, Ice R, Hertel N. 1997. Biokinetic model for ^{137}Cs in the fetus. Health Phys 73(5):736-746.

*CDC. 2001. National report on human exposure to environmental chemicals. Atlanta, GA: National Center for Environmental Health: Centers for Disease Control and Prevention.

Chaplygin VI, Zorov NB, Kuzyakov YY. 1983. Laser atomic-ionization determination of caesium in flames. Talanta 30(7):505-508.

Chernyshov VP, Vykhovanets EV, Slukvin II, et al. 1997. Analysis of blood lymphocyte subsets in children living on territory that received high amounts of fallout of Chernobyl accident. Clin Immunol Immunopathol 84(2):122-128.

Cherouati DE, Djeffal S. 1988. Measurements of radon and radon daughters in dwellings in Algiers. Radiat Prot Dosim 25:137-139.

Chesnokov AV, Fedin VI, Govorun AP, et al. 1997. Collimated detector technique for measuring a 137Cs deposit in soil under a clean protected layer. Appl Radiat Isot 48:1265-1272.

Chiasson AG. 1990. Interference indetermination of rubidium and cesium in fish and zooplankton by graphite furnace atomic absorption spectrometry. J Chem Ecol 16(8):2503-2510.

*Chiavarini S, Galletti M, Michetti I, et al. 1994. Environmental monitoring at Terra Nova Bay Station from 1989-1991. Int J Environ Anal Chem 55:331-340.

Chibowski S, Mitura A. 1995. Studies of the rate of migration of radiocesium in some types of soils of Eastern Poland. Sci Total Environ 170:193-198.

Chillrud SN, Bopp RF, Simpson HJ, et al. 1999. Twentieth century atmospheric metal fluxes into Central Park Lake, New York City. Environ Sci Technol 33(5):657-662.

9. REFERENCES

*Chutke NT, Ambulkar MN, Garg AN. 1995. An environmental pollution study from multielemental analysis of pedestrian dust in Nagpur City, Central India. Sci Total Environ 164:185-194.

Clemente GF, Mariani A, Santaroni GP. 1971. Sex differences in Cs metabolism in man. Health Phys 21:709-711.

*Clewell HJ III, Andersen ME. 1985. Risk assessment extrapolations and physiological modeling. Toxicol Ind Health 1(4):111-131.

Cochran KW, Doull J, Mazur M, et al. 1950. Acute toxicity of zirconium, columbium, strontium, lanthanum, cesium, tantalum and yttrium. Arch Ind Hyg Occup Med 1:637-650.

Coquery M, Carvalho FP, Azemard S, et al. 2000. Certification of trace and major elements and methylmercury concentrations in a macroalgae (*fucus sp.*) reference material, IAEA-140. Fresenius J Anal Chem 366:792-801.

Cornelis R, Speecke A, Hoste J. 1975. Neutron activation analysis for bulk and trace elements in urine. Anal Chim Acta 78:317-327.

Cristaldi M, D'Arcangelo ED, Ieradi LA, et al. 1990. ^{137}Cs determination and mutagenicity tests in wild Mus musculus domesticus before and after the Chernobyl accident. Environ Pollut 64:1-9.

Cristaldi M, Ieradi LA, Mascanzoni D, et al. 1991. Environmental impact of the Chernobyl accident: mutagenesis in bank voles from Sweden. Int J Radiat Biol 59(1):31-40.

Croudace IW, Cundy AB. 1995. Heavy metal and hydrocarbon pollution in recent sediments from Southampton water, Southern England: A geochemical and isotopic study. Environ Sci Technol 29:1288-1296.

Cryer MA, Baverstock KF. 1972. Biological half-life of ^{137}Cs in man. Health Phys 23(3):394-395.

Cuddihy RG, Ozog JA. 1973. Nasal absorption of CsCl, $SrCl_2$, $BaCl_2$, $CeCl_2$ in Syrian hamsters. Health Phys 25:219-224.

Cuddihy RG, Finch GL, Newton GJ, et al. 1989. Characteristics of radioactive particles released from the Chernobyl nuclear reactor. Environ Sci Technol 23:89-95.

Cunningham WC, Anderson DL, Baratta EJ. 1994. Radionuclides in domestic and imported foods in the United States, 1987-1992. J AOAC Int 77(6):1422-1427.

Cunningham WC, Stroube WB, Baratta EJ. 1989. Chemical contaminants monitoring: Radionuclides in domestic and imported foods in the United States, 1983-1986. J Assoc Off Anal Chem 72(1):15-18.

Daburan F, Fayart G, Tricaud Y. 1989. Caesium and iodine metabolism in lactating cows under chronic administration. Sci Total Environ 85:253-261.

da Cruz AD, Glickman BW. 1997. Nature of mutation in the human HPRT gene following in vivo exposure to ionizing radiation of cesium-137. Environ Mol Mutagen 30:385-395.

da Cruz AD, Curado MP, Glickman BW. 1995. The spectrum of mutation of the HPRT gene in human T-lymphocytes following in vivo exposure to ionizing radiation of cesium-137. Environ Mol Mutagen 25(Suppl. 25):10.

da Cruz AD, Curry J, Curado MP, et al. 1996. Monitoring HPRT mutant frequency over time in T-lymphocytes of people accidentally exposed to high doses of ionizing radiation. Environ Mol Mutagen 27:165-175.

da Cruz AD, McArthur AG, Silva CC, et al. 1994. Human micronucleus counts are correlated with age, smoking, and cesium137 dose in the Goiania (Brazil) radiological accident. Mutat Res 313:57-68.

Dam K, Bankl H, Mostbeck A. 1988. Radiocesium incorporation in 250 patients deceased within the year following the Chernobyl disaster. Wien Klin Wochenschr 100(7):193-197.

Danetskaya EV, Ershova VP, Ushakova AP, et al. 1970. [The effect of protein content on ^{137}Cs incorporation and resistance of the organism to chronic internal irradiation.] Med Radiol 15(12):25-30. (Russian).

Dang HS, Jaiswal DD, Sharma RC, et al. 1995. Studies on the biological half-lives of three important radionuclides released in nuclear power reactor operations. Health Phys 69(3):400-402.

Das HA, Comans RNJ. 1990. On the limits of low level measurements of ^{137}Cs as a natural radiotracer. J Radioanal Nucl Chem 139(2):287-295.

*Dasch JM, Wolff GT. 1989. Trace inorganic species in precipitation and their potential use in source apportionment studies. Water Air Soil Pollut 43:401-412.

Densmore CL, Luyyge WG, Emadian SM. 1984. Effects of metal ions and chelating agents on in vitro stability of glucocorticoid receptors in brain cytosol. Life Sci 35:2237-2246.

DeWoody JA. 1999. Nucleotide variation in the p53 tumor-suppressor gene of voles from Chernobyl, Ukraine. Mutat Res 439:25-36.

Dierickx PJ. 1980. Urinary gamma-glutamyl transferase as a specific marker for mercury after heavy metal treatment of rats. Toxicol Lett 6:235-238.

Ding-Hua F, Qi-Jun C. 1998. Study on accumulation of ^{137}Cs in aquatic organisms. Nucl Sci Tech 9(3):184-185.

Dinter H, Pang J, Tesch K. 1988. Calculations of doses due to electron-photon stray radiation from a high energy electron beam behind lateral shielding. Radiat Prot Dosim 25:127-131.

Diomond ML, Mackay D, Cornett RJ, et al. 1990. A model of the exchange of inorganic chemicals between water and sediments. Environ Sci Technol 24:713-722.

*Djingova R, Wagner G, Peshev D. 1995. Heavy metal distribution in Bulgaria using Populus nigra 'Italica' as a biomonitor. Sci Total Environ 172:151-158.

*DOE. 1986. A compendium of the environmental measurements laboratory's research projects related to the Chernobyl nuclear accident. New York, NY: Environmental Measurements Laboratory. U.S. Department of Energy. EML-460. DE87-001847.

9. REFERENCES

*DOE. 1992. Chemical contaminants on DOE lands and selection of contaminant mixtures for subsurface science research. Washington, DC: U.S. Department of Energy. DE92-014826.

*DOE. 1996a. Closing the circle on the splitting of the atom: The environmental legacy of nuclear weapons production in the United States and what the department of energy is doing about it. Washington, DC: Office of Environmental Management. U.S. Department of Energy. DOE/EM-0266.

*DOE. 1996b. Selected radionuclides important to low-level radioactive waste management: National low-level waste management program. U.S. Department of Energy. DOE/LLW-238.

*DOE. 1998a. Heavy element radionuclides (Pu, Np, U) and ^{137}Cs in soils collected from the Idaho national engineering and environmental laboratory and other sites in Idaho, Montana, and Wyoming. New York, NY: Environmental Measurements Laboratory. U.S. Department of Energy. EML-599.

*DOE. 1998b. Savannah river site environmental report for 1998. Aiken, SC: U.S. Department of Energy. WSRC-TR-98-00312. DE-ACO9-96SR18500.

*Doggett NA, McKenzie WH. 1983. An analysis of the distribution and dose response of chromosome aberrations in human lymphocytes after in vitro exposure to ^{137}cesium gamma radiation. Radiat Environ Biophys 22:33-51.

Dokiya T, Wada T, Shi-Long L, et al. 1978. Effect of single irradiation by Californium-252 compared with that of cesium-137 on skins of C3H mice. Nippon Igaku Hoshasen Gakkai Zasshi 38(6):570-576.

*Dombovári J, Becker JS, Kuhn AJ, et al. 2000. Multielement analysis of small plant tissue samples using inductively coupled plasma mass spectrometry. Atom Spectrosc 21(2):37-41.

Domby AH, Paine D, McFarlane RW. 1977. Radiocesium dynamics in herons inhabiting a contaminated reservoir system. Health Phys 33:523-532.

*Dreizen S, Levy BM, Niedermeier W, et al. 1970. Comparative concentrations of selected trace metals in human and marmoset saliva. Arch Oral Biol 15:179-188.

Drichko VF, Popov DK. 1971. Age-related speed of elimination of Cs^{137} from the human body. Med Radiol 16(6):55-57.

Dua SK, Maniyan CG, Kotrappa P. 1987. Inhalation exposures during operations in spent fuel bays. Radiat Prot Dosim 19(3):165-172.

*Ducousso R, Causse A, Pasquier C. 1975. Comparative effects of acetazolamide and Prussian blue on ^{137}Cs retention in the rat. Health Phys 28:75-78.

*Eaman M. 1986. Transfer of radionuclides from the environment to human milk - a review. Ottawa, Canada: Atomic Energy Control Board. DE88705117.

Edelson A, Gottesfeld Z, Samuel D, et al. 1976. Effect of lithium and other alkali metals on brain chemistry and behavior: II. Intracranial self-stimulation behavior. Psychopharmacologia 45:233-237.

*Eisenbud M, ed. 1987. Environmental radioactivity: From natural, industrial, and military sources. New York: Academic Press, Inc.

*Eisler R. 1995. Ecological and toxicological aspects of the partial meltdown of the Chernobyl nuclear power plant reactor. In: Hoffman DJ, Rattner BA, Burton GAJ, et al., ed. Handbook of Ecotoxicology. Tokyo: Lewis Publishers, 15.

Elder JF, Collins JJ. 1991. Freshwater molluscs as indicators of bioavailability and toxicity of metals in surface-water systems. Rev Environ Contam Toxicol 122:37-79.

Eliseeva KN, Kartel NA, Voitovich AM, et al. 1996. Chromosomal aberrations in different tissues of rodents and amphibians from the regions of Belarus contaminated by radionuclides. Tsitol Genet 30(4):20-25.

*Ellenhorn MJ, Schonwald S, Ordog G, et al. 1997. Medical toxicology: Diagnosis and treatment of human poisoning. 2nd ed. Baltimore: Williams & Wilkins. 1682-1723.

EMMI. 1992. EML Procedures Manual, 27th ed. Vol 1. Environmental Measurements Laboratory. U.S. Department of Energy. Environmental Methods Monitoring Index.

*EPA. 1980. Prescribed procedures for measurement of radioactivity in drinking water. Cincinnati, OH: Office of Research and Development. U.S. Environmental Protection Agency. EPA-600/4-80-032.

*EPA. 1989. Environmental radiation data. Report 58. Montgomery, AL: Office of Radiation Programs. U.S. Environmental Protection Agency. EPA 520/5-89-034.

*EPA. 1990a. Characterization of municipal waste combustion ash, ash extracts, and leachates. Solid Waste and Emergency Response. U.S. Environmental Protection Agency. EPA 530-SW-90-029A.

*EPA. 1990b. Interim methods for development of inhalation reference concentrations. Washington, DC: Office of Health and Environmental Assessment. Office of Research and Development. Environmental Criteria and Assessment Office. U.S. Environmental Protection Agency. EPA 600/8-90/066A.

*EPA. 1997a. Special report on environmental endocrine disruption: An effects assessment and analysis. Washington, DC: Risk Assessment Forum. U.S. Environmental Protection Agency. EPA/630/R-96/012.

*EPA. 1997b. Health effects assessment summary tables. Washington, DC: Office of Emergency and Remedial Response. Office of Research and Development. U.S. Environmental Protection Agency. EPA 540/R-97-036. PB97-921199.

*EPA. 1998. Ionizing radiation series no. 1. Office of Radiation and Indoor Air Radiation Protection Division. U.S. Environmental Protection Agency. EPA 402-F-98-009. Http://www.epa.gov/radiationionize.htm. October 11, 2000.

*EPA. 1999a. Compliance procedures method for determining compliance with subpart I. U.S. Environmental Protection Agency. 40 CFR 61 App E.

*EPA. 1999b. Analytical methods for radioactivity. U.S. Environmental Protection Agency. 40 CFR 141.25.

*EPA. 1999c. Offsite environmental monitoring report: Radiation monitoring around United States nuclear test areas, calender year 1997. Washington, DC: Office of Radiation and Indoor Air. U.S. Environmental Protection Agency. EPA 402/R-98-013.

Erkin VG, Lebedev OV. 1993. Thermoluminescent dosimeter measurements of external doses to the population of the Bryansk region after the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 289-311.

Erten HN, Aksoyoglu S, Gokturk H. 1988. Sorption, desorption of Cs on clay and soil fractions from various regions of Turkey. Sci Total Environ 69:269-296.

Evets LV, Lyalikov SA, Orekhova SD, et al. 1992. The biological effect of low-level radiation on morphological composition. Radiobiologiia 32(5):627-631.

*Eyman LD, Kevern NR. 1975. Cesium-137 and stable cesium in a hypereutrophic lake. Health Phys 28:549-555.

Fabbris R, Gonella F, De Angelis A, et al. 1988. Environmental large-scale monitoring of ^{137}Cs activity by milk contamination measurements and resultant doses. Radiat Prot Dosim 25:141-142.

Farkas RH, Chien P-Y, Nakajima S, et al. 1996. Properties of a slow nonselective cation conductance modulated by neurotensin and other neurotransmitters in midbrain dopaminergic neurons. J Neurophysiol 76(3):1968-1981.

Fedorova A, Yakovleva N, Nechev K. 1975. Effect of isolated radionuclides on the hematolytic and biochemical blood indexes. Rentgenol Radiol 14(2):108-112.

*FEDRIP. 2000. Federal Research in Progress. Dialog Information Services, Inc. Palo Alto, CA.

*Feely HW, Helfer IK, Juzdan ZR, et al. 1988. Fallout in the New York metropolitan area following the Chernobyl accident. J Environ Radioact 7:177-191.

Fehlings MG, Nashmi R. 1996. Changes in pharmacological sensitivity of the spinal cord to potassium channel blockers following acute spinal cord injury. Brain Res 736:135-145.

*Feldman C, Rains TC. 1964. The collection and flame photometric determination of cesium. Anal Chem 36:405-409.

*Fernandez MA, Martinez L, Segarra M, et al. 1992. Behavior of heavy metals in the combustion gases of urban waste incinerators. Environ Sci Technol 26(5):1040-1047.

*Fernandez-Turiel JL, Lopez-Soler A, Llorens JF, et al. 1995. Environmental monitoring using surface water, river sediments, and vegetation: A case study in the Famatina range, La Rioja, NW Argentina. Environ Int 21(6):807-820.

Feuger GF. 1986. Biological effects and risks of accidental radionuclide uptake. Acta Med Austriaca 13:86-93.

Fields DE, Yalcintas MG. 1989. Lifestyle and age as determinates of intake and dose from inhaled radionuclides. J Radiol Prot 9(4):251-255.

*Fisher NS, Fowler SW, Boisson F, et al. 1999. Radionuclide bioconcentration factors and sediment partition coefficients in arctic seas subject to contamination form dumped nuclear wastes. Environ Sci Technol 33(12):1979-1982.

*Fomon SJ. 1966. Body composition of the infant: Part I: The male "reference infant." In: Falkner F, ed. Human development. Philadelphia, PA: WB Saunders, 239-246.

*Fomon SJ, Haschke F, Ziegler EE, et al. 1982. Body composition of reference children from birth to age 10 years. Am J Clin Nutr 35:1169-1175.

Fowler SW. 1986. Trace metal monitoring of palagic organisms from the open Mediterranean Sea. Environ Monit Assess 7:59-78.

Francois A, Aigueperse J, Gourmelon P, et al. 1998. Exposure to ionizing radiation modifies neurally-evoked electrolyte transport and some inflammatory responses in rat colon in vitro. Int J Radiat Biol 73(1):93-101.

Frantsevich L, Korniushin A, Pankov I, et al. 1996. Application of molluscs for radioecological monitoring of the Chernobyl outburst. Environ Pollut 94(1):91-100.

Frenzilli G, Lori A, Panasiuk G, et al. 1998. Comet assay on children's leukocytes 8 years after the Chernobyl disaster. Mutat Res 415:151-158.

*Fresquez PR, Armstrong DR, Mullen MA. 1998. Radionuclides in soils collected from within and around Los Alamos National Laboratory: 1974-1996. J Environ Sci Health Part A 33(2):263-278.

*Fresquez PR, Biggs JR, Bennett KD, et al. 1999a. Radionuclides in deer and elk from Los Alamos National Laboratory and the doses to humans from the ingestion of muscle and bone. J Environ Sci Health B 34(5):901-915.

Fresquez PR, Kraig DH, Mullen MA, et al. 1999b. Radionuclides and trace elements in fish collected upstream and downstream of Los Alamos National Laboratory and the doses to humans from the consumption of muscle and bone. J Environ Sci Health B 34(5):885-889.

*Frigieri P, Trucco R, Ciaccolini In, et al. 1980. Determination of caesium in river and seawaters by electrothermal atomic-absorption spectrometry. Interference of cobalt and iron. Analyst 105:651-656.

Fuchs VR. 1981. Low-level radiation and infant mortality. Health Phys 40:847-854.

Fujita M, Takata K, Iwamoto J. 1968. Retention, excretion and blood concentration of ^{137}Cs by rat as influenced by starvation. J Radiat Res 9:129-134.

Fujita M, Yabe A, Akaishi J, et al. 1966. Relationship between ingestion, excretion and accumulation of fallout cesium-137 in man on long-term scale. Health Phys 12:1649-1653.

*Fülöp M, Ragan P. 1997. *In-situ* measurements of ^{137}Cs in soil by unfolding method. Health Phys 72:923-930.

Furchner JE, Richmond CR, Drake GA. 1965. Effects of environmental temperature on retention of chronically administered cesium-137. Health Phys 11:623-628.

*Furchner JE, Trafton GA, Richmond CR. 1964. Distribution of cesium[137] after chronic exposure in dogs and mice. Proc Soc Exp Biol Med 116:375-378.

Ganzerli Valentini MI, Stella R, Maggi L, et al. 1987. Copper hexacyanoferrate(II) and (III) as trace cesium absorbers from natural waters. J Radioanal Nucl Chem 114(1):105-112.

Garnier J-M, Pham MK, Ciffroy P, et al. 1997. Kinetics of trace element complexion with suspended matter and with filterable ligands in freshwater. Environ Sci Technol 31:1597-1606.

*Gaur S. 1996. Determination of Cs-137 in environmental water by ion-exchange chromatography. J Chromatog A733:57-71.

*Gautier MA. 1983. Manual of analytical methods for radiobioassay. Los Alamos, NM: Los Alamos National Laboratory. LA-9763-M Manual.

Gawlik D, Behne D, Kraft D, et al. 1989. The influence of renal insufficiency on caesium metabolism in man and rat (with a note in the Cs content of some biological standard materials). J Trace Elem Electrolytes Health Dis 3:43-50.

Genicot J-L. 1997. Room-temperature semiconductor detectors for *in vivo* monitoring of internal contamination. Environ Health Perspect 105(Suppl. 6):1423-1426.

*Gerber GB, Thomas RG. 1992. Guidebook for the treatment of accidental internal radionuclide contamination of workers. Radiat Prot Dosim 41:1-49.

*Ghosh A, Sharma A, Talukder G. 1990. Clastogenic effects of cesium chloride on mouse marrow cells in vivo. Mutat Res 244:295-298.

*Ghosh A, Sharma A, Talukder G. 1991. Cytogenetic damage induced in vivo to mice by single exposure to cesium chloride. Environ Mol Mutagen 18:87-91.

*Ghosh A, Sharma A, Talukder G. 1993. Clastogenic effects of caesium chloride on human peripheral blood lymphocytes in vitro. Toxicol in Vitro 7(2):137-140.

Ginsborg BL, Hamilton JT. 1968. The effect of caesium ions on neuromuscular transmission in the frog. Q J Exp Physiol 53:162-169.

Gitterman M, Webster EW. 1984. Shielding hospital rooms for brachytherapy patients: Design, regulatory and cost/benefit factors. Health Phys 46(3):617-625.

Giusti L, Yang Y-L, Hewitt CN, et al. 1993. The solubility and partitioning of atmospherically derived trace metals in artificial and natural waters: A review. Atmos Environ 27A(10):1567-1578.

*Giwercman A, Carlsen E, Keiding N, et al. 1993. Evidence for increasing incidence of abnormalities of the human testis: A review. Environ Health Perspect Suppl 101(2):65-71.

*Glowiak BJ, Pacyna J, Halawa B. 1977a. Contamination of individual human organs by strontium-90 and cesium-137. Nachr Mensch-Umwelt 5(4):12-29.

*Glowiak BJ, Pacyna J, Palczynski RJ. 1977b. Strontium and caesium-137 contents in human teeth. Environ Pollut 14:101-111.

Gluzman DF, Simonet ML, Moutet A, et al. 1994. Ionizing radiation induced leukemias. Eksp Onkol 16:83-95.

Gnedko TV. 1995. Blood indices in newborns and their mothers from Belarus districts contaminated with radionuclides after the Chernobyl accident. Biorheology 32:403.

Golikov VY, Balonov MI, Ponomarev AV. 1993. Estimation of external gamma radiation doses to the population after the Chernobyl accident. In: Merwin SE, Balonov MI, ed. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 247-288.

*Gomes MA, Wascheck CC, Scully C, et al. 1990. Orofacial manifestations from accidental exposure to caesium 137 in Goiania, Brazil. J Oral Pathol Med 19(7):322-325.

Gone JK, Olmez I, Ames MR. 2000. Size distribution and probable sources of trace elements in submicron atmospheric particulate material. J Radioanal Nucl Chem 244:133-139.

Goulko GM, Chepurny NI, Jacob P, et al. 1998. Thyroid dose and thyroid cancer incidence after the Chernobyl accident: Assessments for the Zhytomyr region (Ukraine). Radiat Environ Biophys 36:261-273.

Grobenski Z, Weber D, Welz B, et al. 1983. Determination of caesium and rubidium by flame and furnace atomic-absorption spectrometry. Analyst 108:925-932.

Grovoba OM, Chernikov VP. 1996. [The occurrence of 137Cs in the tissue of pulmonary tumor in a man exposed to radiation after the Chernobyl incident.] Ter Arkh 68(3):26-30. (Russian).

Gupta MM, Jain SC, Nagaratnam A. 1998. Effective dose estimates for Indian adults from ingestion of ^{3}H, ^{131}I and ^{137}Cs. Radiat Prot Dosim 79:287-288.

*Guzelian PS, Henry CJ, Olin SS, eds. 1992. Similarities and differences between children and adults: Implications for risk assessment. Washington, DC: International Life Sciences Institute Press.

*Haddad LM, Shannon MW, Winchester JF, eds. 1998. Clinical management of poisoning and drug overdose. 3rd ed. Philadelphia: WB Saunders. 413-425.

Hahn FF, McClellan RO, Benjamin SA, et al. 1977a. Toxicity of inhaled beta-emitting radionuclides - an experimental approach. In: Annual report of the inhalation toxicology research institute. Albuquerque: Inhalation Toxicology Research Institute, 149-153.

Hahn FF, McClellan RO, Benjamin SA, et al. 1977b. Toxicity studies of inhaled beta-emitting radionuclides - an experimental approach. In: Annual report of the inhalation toxicology research institute. Albuquerque: Inhalation Toxicology Research Institute, 57-61.

Hahn FF, McClellan RO, Boecker BB, et al. 1979. Toxicity studies of inhaled beta-emitting radionuclides - status report. In: Annual report of the inhalation toxicology research institute. Albuquerque: Inhalation Toxicology Research Institute, 51-56.

*Hakem N, Mahamid A, Apps J, et al. 1997. Sorption of cesium and strontium on Savannah river soils impregnated with colloidal silica. In: Conference proceedings - International containment technology conference. Springfield, VA: National Technical Information Service, 652-657.

*Hakonson TE, Whicker FW. 1975. Cesium kinetics in a montane lake ecosystem. Health Phys 28:699-706.

Halbrook RS, Jenkins JH. 1988. Cesium-137 levels detected in Georgia otters. Bull Environ Contam Toxicol 41:765-769.

Hammond SE, Bold FO, Macdonald NS. 1963. Cesium-137 excretion and retention following single exposure. Health Phys 9:523-528.

Hanich RF, Levine JH, Spear JF, et al. 1988. Autonomic modulation of ventricular arrhymia in cesium chloride-induced long GT syndrome. Circulation 77(5):1149-1161.

Hanika-Rebar C, Rebar AH, Boecker BB, et al. 1978. Toxicity of injected ^{137}CsCl in beagle dogs. XI. In: McClellan RO, ed. Annual report of the inhalation toxicology research institute. Albuquerque: Lovelace Biomedical and Environmental Research Institute, 80-83.

Hanlon DP, Ferm VH. 1986. Teratogen concentration changes as the basis of the heat stress enhancement of arsenate teratogenesis in hamsters. Teratology 34:189-193.

Hanna LA, Peters JM, Wiley LM, et al. 1997. Enhancing effect of maternal zinc deficiency and ^{137}Cs γ-irradiation on the frequency of fetal malformations in mice. Teratog Carcinog Mutagen 17:127137.

Harjulehto T, Aro T, Rita H, et al. 1989. The accident at Chernobyl and outcome of pregnancy in Finland. Br Med J 298:995-997.

Harrison JD. 1995. Ingested radionuclides. In: Potten CS, Hendry JH, eds. Radiation and gut. New York, NY: Elsevier Science Publishers, 253-289.

Harrison SE, Klaverkamp JF, Hesslein RH. 1990. Fates of metal radiotracers added to a whole lake: Accumulation in fathead minnow (Pimephales promelas) and lake trout (Salvelinus namaucush). Water Air Soil Pollut 52:277-293.

Havlicek F. 1968. Metabolism of radiocesium during gestation and lactation as influenced by ferric-cyanoferrate(II). Int J Appl Radiat Isot 19:487-488.

Hawthorne HA, Zellmer SD, Eberhardt LL, et al. 1976. ^{137}Cesium cycling in a Utah dairy farm. Health Phys 30:447-464.

Haynes RM. 1988. The distribution of domestic radon concentrations and lung cancer mortality in England and Whales. Radiat Prot Dosim 25:93-96.

HazDat. 2000. Cesium. ATSDR's hazardous substance release and health effects database. Agency for Toxic Substances and Disease Registry. Http://www.atsdr.cdc.gov/hazdat.html. August 29, 2000.

*HazDat. 2001. Atlanta, GA: Agency for Toxic Substances and Disease Registry (ATSDR). Http://www.atsdr.cdc.gov/gsql/getsubstance.script?in*cas=007440-46-2*. April 23, 2001.

Hellman S. 1977. The place of radiation in the definitive management of carcinoma of the breast. Int J Radiat Oncol Biol Phys 2:369-370.

*Hellou J, Fancey LL, Payne JF. 1992a. Concentrations of twenty-four elements in bluefin tuna, *thunnus thynnus* from the Northwest Atlantic. Chemosphere 24(2):211-218.

Hellou J, Warren WG, Payne JF, et al. 1992b. Heavy metals and other elements in three tissues of cod, Gadus morhua from the Northwest Atlantic. Mar Pollut Bull 24(9):452-458.

*Helmers E. 1996. Elements accompanying platinum emitted from automobile catalysts. Chemosphere 33(3):405-419.

*Henrichs K, Paretzke HG, Voigt G, et al. 1989. Measurements of Cs absorption and retention in man. Health Phys 57(4):571-578.

Herman P, Tietveld EC, Weseman JM. 1987. The metabolism and tissue accumulation of radio-cesium in veal calves throughout the entire fattening period. J Anim Physiol Anim Nutr 60:108-112.

*Hewitt PJ. 1988. Accumulation of metals in the tissues of occupationally exposed workers. Environ Geochem Health 10:113-116.

*Hillmann U, Schimmack W, Jacob P, et al. 1996. In situ γ-spectrometry several years after deposition of radiocesium: Part I: Approximation of depth distributions by the Lorentz function. Radiat Environ Biophys 35:297-303.

*Hilton J, Livens FR, Spezzano P, et al. 1993. Retention of radioactive caesium by different soils in the catchment of a small lake. Sci Total Environ 129:253-266.

*Hintenlang DE. 1993. Synergistic effects of ionizing radiation and 60 Hz magnetic fields. Bioelectromagnetics 14:545-551.

*Hirose K, Takatani S, Aoyama M. 1993. Wet deposition of radionuclides derived from the Chernobyl accident. J Atmos Chem 17:61-71.

*Hoel DG, Davis DL, Miller AB, et al. 1992. Trends in cancer mortality in 15 industrialized countries, 1969-1986. J Natl Cancer Inst 84(5):313-320.

Hood SL, Comar CL. 1953. Metabolism of cesium-137 in rats and farm animals. Arch Biochem Biophys 45:423-433.

Hoshi M, Shibata Y, Okajima S, et al. 1994a. ^{137}Cs concentration among children in areas contaminated with radioactive fallout from the Chernobyl accident: Mogilev and Gomel Oblasts, Belarus. Health Phys 67(3):272-275.

Hoshi M, Yamamoto M, Kawamura H, et al. 1994b. Fallout radioactivity in soil and food samples in the Ukraine: Measurements of iodine, plutonium, cesium, and strontium isotopes. Health Phys 67(2):187-191.

Huang C-Y, Lee J-D, Tseng C-L, et al. 1994. A rapid method for the determination of ^{137}Cs in environmental water samples. Anal Chim Acta 294:221-226.

*Huda M, Sourkes AM, Tracy BL. 1988. Chernobyl - The radiological impact on Canada. J Can Assoc Radiol 39:37-41.

9. REFERENCES

*IAEA. 1962. Whole-body counting. Vienna: International Atomic Energy Agency. IAEA Publication No. STI/PUB/47.

*IAEA. 1970. Directory of whole-body radioactivity monitors. Vienna: International Atomic Energy Agency. IAEA Publication No. STI/PUB/213.

*IAEA. 1972. Assessment of radioactive contamination in man. Vienna: International Atomic Energy Agency. IAEA Publication No. STI/PUB/290.

*IAEA. 1976. Diagnosis and treatment of incorporated radionuclides. Vienna: International Atomic Energy Agency. IAEA Publication No. STI/PUB/411.

*IAEA. 1985. Assessment of radioactive contamination in man 1984. Vienna: International Atomic Energy Agency. IAEA Publication No. STI/PUB/674.

*IAEA. 1988. The radiological accident in Goiania. Vienna: International Atomic Energy Agency. IAEA Publication No. STI/PUB/815.

*IAEA. 1991. The international Chernobyl project technical report, page 2 of 7. International Atomic Energy Agency, Vienna. Publication No. STI/PUB/885.

*IARC. 2000. Cesium. International Agency for Research on Cancer. Http://193.51.164.11.html. October 13, 2000.

ICRP. 1975. Report of the task group on reference man. International Commission on Radiological Protection. Oxford: Pergamon Press: ICRP Publication 23.

*ICRP. 1979. Limits for intakes of radionuclides by workers. International Commission on Radiological Protection. Oxford: Pergamon Press. ICRP Publication 30, Part 1; Annuals of the ICRP 2(3/4).

*ICRP. 1983. Radionuclide transformations: Energy and intensity of emissions. International Commission on Radiological Protection. Oxford: Pergamon Press. ICRP publication 38. 484-503.

*ICRP. 1989. Age dependent doses to members of the public from intake of radionuclides. International Commission on Radiological Protection. Oxford: Pergamon Press. ICRP Publication 56, Part 1; Annuals of the ICRP 20(2).

*ICRP. 1993. Age dependent doses to members of the public from intake of radionuclides. Part 2; Ingestion dose coefficients. International Commission on Radiological Protection. Oxford: Pergamon Press. ICRP Publication 67, Part 1; Annuals of the ICRP 23(3/4).

*ICRP. 1994. Human respiratory tract model for radiological protection. International Commission on Radiological Protection. Pergamon Press, Oxford. Publication No. 66.

*ICRP. 1995. Age-dependent doses to members of the public from intake of radionuclides: Part 4 inhalation dose coefficients. Pergamon Press, Oxford. Publication No. 71.

ICRP. 1999. Protection of the public in situations of prolonged radiation exposure. International Commission on Radiological Protection. Pergamon Press, Oxford. Publication No. 82

*Ide HM, McInroy JF. 1975. Cesium-134 in human autopsy tissue. Health Phys 29:871-872.

*Iijima K, Morimoto K. 1991. Quantitative analysis of the induction of chromosome aberrations and sister-chromatid exchanges in human lymphocytes exposed to γ-rays and mitomycin-C in combination. Mutat Res 263:263-268.

*Iinuma T, Nagai T, Ishihara T. 1965. Cesium turnover in man following single administration of 132Cs: 1. Whole body retention and excretion pattern. J Radiat Res 6:73-81.

Ilyin LA, Ivannikov AT, Popov BA, et al. 1998. [Study of ^{137}Cs transfer regularities through the placenta to the rat fetuses and the effect of ferrocinum on these processes.] Radiat Biol Radioecol 38(4):616-623. (Russian).

Ilyinskikh NN, Ilyinskikh EN, Ilyinskikh IN. 1998. Micronucleated erthrocytes frequency and radiocesium bioconcentration in pikes (Esox lucius) caught in the Tom River near the nuclear facilities of the Siberian chemical complex (Tomsk-7). Mutat Res 421:197-203.

Ilyinskikh NN, Ilyinskikh IN, Smirnov BV, et al. 1999. Micronuceated erthrocytes and radionuclide activity in persons living in a radiation contaminated area near the Mayak nuclear facility (Russia). J Hum Ecol 10(4):237-244.

Inaba J, Matsusaka N, Ichikawa R. 1967. Whole-body retention and tissue distribution of cesium-134 in new-born, young and adult rats. J Radiat Res 8:132-140.

Inalsingh CHA, Chandrasekaran MS, Julian C, et al. 1976. A seven year review of invasive carcinoma of the cervix treated at the Johns Hopkins Hospital. Johns Hopkins Med J 139:147-151.

*IRIS. 2000. Cesium. U.S. Environmental Protection Agency. Integrated Risk Information System. Http://www.epa.gov/iris/subst/index.htm. October 24, 2000.

*Isenberg G. 1976. Cardiac purkinje fibers: Cesium as a tool to block inward rectifying potassium currents. Pflugers Arch 365:99-106.

Ito M, Yamashita S, Ashizawa K, et al. 1996. Histopathological characteristics of childhood thyroid cancer in Gomel, Belarus. Int J Cancer 65:29-33.

*Iyengar V, Woittiez J. 1988. Trace elements in human clinical specimens: Evaluation of literature data to identify reference values. Clin Chem 34:474-481.

Jenner FA, Judd A, Parker J. 1975. The effects of lithium, rubidium and caesium on the response of rats to tranylcypromine and alpha-methyl-p-tyrosine given separately or in combination. Br J Pharmacol 54:233-234.

Jin H, Gao L, Xu Z, et al. 1996. Effect of low dose irradiation on nutrition metabolism in rats. Yingyang Xuebao 18(2):134-138.

*Johanson CE. 1980. Permeability and vascularity of the developing brain: Cerebellum vs cerebral cortex. Brain Res 190:3-16.

*Johansson L, Bjoreland A, Agren G. 1998. Transfer of ^{137}Cs to infants via human breast milk. Radiat Prot Dosim 79:165-167.

*Johnson FN. 1972. Effects of alkali metal chlorides on activity in rats. Nature 238:333-334.

*Johnson GT, Lewis TR, Wagner WD. 1975. Acute toxicity of cesium and rubidium compounds. Toxicol Appl Pharmacol 32:239-245.

Jouve A, Lejeune M, Rey J. 1999. A new method for determining the bioavailability of radionuclides in the soil solution. J Environ Radioact 43:277-289.

Kahn B, Smith DK, Straub CP. 1957. Determination of low concentrations of radioactive cesium in water. Anal Chem 29:1210-1213.

Kali AK, Whitmore R, Roache W. 1985. Behavior of cesium in fish tissues from Savannah River. Environ Int 11:23-24.

*Kamiguchi Y, Tateno H, Mikamo K. 1991. Micronucleus test in 2-cell embryos as a simple assay for human sperm chromosome aberrations. Mutat Res 252:297-303.

*Kanapilly GM, Stanley JA, Newton GJ, et al. 1983. Characterization of an aerosol sample from the auxillary building of the Three Mile Island reactor. Health Phys 45:981-989.

Kasatkani EP, Shilin DE, Rosenbloom AL, et al. 1997. Effects of low level radiation from the chernobyl accident in a population with iodine deficiency. Eur J Pediatr 156:916-920.

Kashparov VA, Protsak VP, Yoschenko VI, et al. 1994. Inhalation of radionuclides during agricultural work in areas contaminated as a result of the Chernobyl reactor accident. J Aerosol Sci 25(5):761-766.

Kennish MJ. 1998. Trace metal-sediment dynamics in estuaries: Pollution assessment. Rev Environ Contam Toxicol 155:69-110.

*Khosid GM. 1967. ЦЕЗИЙ И РУБИДИЙ. In: Israelson AL, ed. Novye dannye po toksikologii redkikh metalovi. Moscow. 56-63.

*Kim EY, Goto R, Tanabe S, et al. 1998. Distribution of 14 elements in tissues and organs of oceanic seabirds. Arch Environ Contam Toxicol 35:638-645.

Kirchner G. 1994. Transport of iodine and cesium via the grass-cow-milk pathway after the Chernobyl accident. Health Phys 66(6):653-665.

Kirchner TB, Whicker FW, Anspaugh LR, et al. 1996. Estimating internal dose due to ingestion of radionuclides from Nevada test site fallout. Health Phys 71(4):487-501.

Kirillov SA, Vissonov YV. 1971. Clinical picture of Cs^{137} and Cs^{134} poisoning during long-term periods. Med Radiol 16(11):47-52.

Koenig W. 1974. Significance of radiation hazard for the fixing of radiation protection limits. Rep - Staatl Zent Strahlenschutz DDR 157:38-41.

Koike K, Yabuhara A, Yang F-C, et al. 1995. Frequent natural killer cell abnormality in children in an area highly contaminated by the Chernobyl accident. Int J Hematal 61:139-145.

*Kolb W. 1971. Tungsten-181 and other short lived fission products in ground level air in north Germany and north Norway. Nature 232:552-553.

*Komori M, Nishio K, Kitada M, et al. 1990. Fetus-specific expression of a form of cytochrome P-450 in human livers. Biochemistry 29:4430-4433.

Konings J, Kirchmann R, Gerber GB, et al. 1984. Distribution of ^{60}Co and ^{137}Cs in mallard ducks (Anas platyrhynchus). Health Phys 46(3):681-684.

Konshin OV. 1992. Transfer of ^{137}Cs from soil to grass-analysis of possible sources of uncertainty. Health Phys 63(3):307-315.

Korky JK, Kowalski L. 1989. Radioactive cesium in edible mushrooms. J Agric Food Chem 37:568-569.

*Korobova E, Ermakov A, Linnik V. 1998. ^{137}Cs and ^{90}Sr mobility in soils and transfer in soil-plant systems in the Novozybkov district affected by the Chernobyl accident. Appl Geochem 13(7):803-814.

Korolev GK. 1965. Distribution of Cs^{137} after endotracheal administration. Fed Proc 25(1):95-98.

*Koshimoto C, Takahashi S, Kubota Y, et al. 1994. Evaluation of the effect of gamma-irradiation on fetal erythropoiesis in rats using blood cell volume as the index. J Radiat Res 35:74-82.

Kostial K, Vnucec M, Tominac C, et al. 1980. A method for a simultaneous decrease of strontium, caesium and iodine retention after oral exposure in rats. Int J Radiat Biol 37(3):347-350.

Koulikov AO, Ryabov IN. 1992. Specific cesium activity in freshwater fish and the size effect. Sci Total Environ 112:125-142.

Krachler M, Rossipal E, Micetic-Turk D. 1999. Concentrations of trace elements in arterial and venous umbilical cord sera. Trace Elem Electrolytes 16(1):46-52.

Kraemer SM, Waldren CA. 1997. Chromosomal mutations and chromosome loss measured in a new human-hamster hybrid cell line, A_LC: studies with colcemid, ultraviolet irradiation, and ^{137}Cs γ-rays. Mutat Res 379:151-166.

Kraybill HF. 1983. Assessment of human exposure and health risk to environmental contaminants in the atmosphere and water with special reference to cancer. J Environ Sci Health C 1(2):175-232.

*Krieger HL, Martin ER, Frishkorn GW. 1976. Sequential radiochemical analysis for ruthenium, strontium, and cesium in environmental air. Health Phys 30:465-470.

*Krishnan K, Andersen ME. 1994. Physiologically based pharmacokinetic modeling in toxicology. In: Hayes AW, ed. Principles and methods of toxicology. 3rd ed. New York, NY: Raven Press, Ltd., 149-188.

*Krishnan K, Andersen ME, Clewell HJ III, et al. 1994. Physiologically based pharmacokinetic modeling of chemical mixtures. In: Yang RSH, ed. Toxicology of chemical mixtures: Case studies, mechanisms, and novel approaches. San Diego, CA: Academic Press, 399-437.

9. REFERENCES

Kryshev II, Romanov GN, Sazykina TG, et al. 1998. Environmental contamination and assessment of doses from radiation releases in the southern Urals. Health Phys 74(6):687-697.

Kubic M, Kusmierek E, Michalczuk L, et al. 1993. Contamination of fruits, soils, and mushrooms in Poland in 1991. J Fruit Ornamental Plant Res 1(2):46-53.

Kudelsky AV, Smith JT, Ovsiannikova SV, et al. 1996. Mobility of Chernobyl-derived ^{137}Cs in a peatbog system within the catchment of the Pripyat River, Belarus. Sci Total Environ 188:101-113.

*Kusama T, Hasegawa Y. 1993. Stage differences in developmental disorders in ICR mouse embryos irradiated with gamma-rays. Congen Anom 33:115-123.

Kwadow M, Chevalier C. 1988. Occupational exposure to radionuclides in French nuclear power plants: Five years excretion monitoring results. Sci Total Environ 70:299-319.

*LaBrecque JL, Rosales PA. 1996. The migration of ^{137}Cs in Venezuelan soils. J Trace Microprobe Tech 14(1):213-221.

*LA Dept Environ Quality. 2000. Radiation protection. Louisiana Department of Environmental Quality. Http://www.deq.state.la.us/. August 29, 2000.

*Landeen DS, Mitchell RM. 1986. Radionuclide uptake by trees at a RAD waste pond in Washington state. Health Phys 50(6):769-774.

Lassy KR. 1979. The transfer of radiostrontium and radiocesium from soil to diet: Models of consistent with fallout analysis. Health Phys 37:557-573.

Lavrent LN. 1975. Nephrosclerosis induced by various chronic radiation effects. Radiobiologiia 15(5):704-707.

Lazjuk GI, Kirillova IA, Nikolaev DL, et al. 1993. Monitoring of congenital malformations in Belarus after the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 385-397.

Lazjuk GI, Nikolaev DL, Novikova IV. 1997. The dynamics of congenital malformations in Belarus Republic. Am J Hum Genet 61(4):A204.

Lebedev OV, Yakovlev VA. 1993a. The correlation between ^{137}Cs half-time and age, body mass and height in individuals contaminated from the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 219-243.

*Leeder JS, Kearns GL. 1997. Pharmacogenetics in pediatrics: Implications for practice. Pediatr Clin North Am 44(1):55-77.

*Leggett RW. 1986. Predicting the retention of Cs in individuals. Health Phys 50(6):747-759.

*Lengemann FW. 1970. ^{134}Cs retention in rats from one to 25 months of age. Radiat Res 42:169-174.

9. REFERENCES

*LeRoy GV, Rust JH, Hasterlik RJ. 1966. The consequences of ingestion by man of real and simulated fallout. Health Phys 12:449-473.

*Leung H-W. 1993. Physiologically-based pharmacokinetic modelling. In: Ballentine B, Marro T, Turner P, eds. General and applied toxicology. Vol 1. New York, NY: Stockton Press, 153-164.

*Levine JH, Spear JF, Guarnieri T, et al. 1985. Cesium chloride-induced long QT syndrome: demonstration of after depolarizations and triggered activity in vivo. Circulation 72(5):1092-1103.

*Lewis RJ. 1997. Hawley's condensed chemical dictionary. 13th ed. New York, NY: John Wiley & Sons. 234-236.

Liaginskaia AM, Osipov VA, Dement'ev SI. 1998. Kinetics of metabolism and mechanisms of formation of absorbed doses in the mouse testis from incorporated Cs^{137}. Radiat Biol Radioecol 38(1):27-30.

Liao M, Wang Z, Yu T. 1988. [Study of 45 metal compounds using micronucleus test]. Weisheng Dulixue Zazhi 2(2):83-86. (Chinese).

*Lide DR. 1998. CRC handbook of chemistry and physics: A ready-reference book of chemical and physical data. 79th ed. Boca Raton: CRC Press. 12/43-12/45.

*Lie R. 1964. Deposition and retention of ^{137}Cs in the rat following inhalation of the chloride and the nitrate. Health Phys 10:1071-1076.

Lievens P, Cornelis R, Hoste J. 1975. A separation scheme for the determination of trace elements in biological materials by neutron activation analysis. Anal Chim Acta 80:97-108.

*Lipsztein JL, Bertilli L, Melo DR, et al. 1991. Application of *in-vitro* bioassay for ^{137}Cs during the emergency phase of the Goiânia accident. Health Phys 60:43-49.

Lipsztein JL, Melo DR, Oliveira CAN, et al. 1998. The Goiania ^{137}Cs accident - a review of the internal and cytogenetic dosimetry. Radiat Prot Dosim 79:149-154.

Livens FR, Horrill AD, Singleton DL. 1991. Distribution of radiocesium in the soil-plant systems of upland areas of Europe. Health Phys 60(4):539-545.

*Livingston, AL. 1978. Forage plant estrogens. J Toxicol Environ Health 4:301-324.

Lloyd DC, Finnon P, Edwards AA, et al. 1997. Chromosome aberrations in Syrian hamsters following very low radiation doses in vivo. Mutat Res 377:63-68.

*Lloyd RD, Mays CW, McFarland SS, et al. 1973. Metabolism of ^{83}Rb and ^{137}Cs in persons with muscle disease. Radiat Res 54:463-478.

Lloyd RD, Zundel WS, Mays CW, et al. 1968. Short caesium half-times in patients with muscular dystrophy. Nature 220:1029-1031.

Lo CK, Fung YS. 1992. Heavy metal pollution profiles of dated sediment cores from Hebe Haven, Hong Kong. Water Res 26(12):1605-1619.

9. REFERENCES

Lockhart WL, Muir CG, Wilkinson P, et al. 1998. Chemical contaminants in fish and sediment core samples from the Dnipro River, Ukraine, 1994. Water Qual Res J Can 33(4):489-509.

*Lokobauer N, Bauman A, Maracic M. 1988. Assessment of the radiation dose in the republic of Croatia after the Chernobyl reactor accident. Environ Int 14:137-143.

Lorenz R, Deubel W, Leuner K, et al. 1994. Dose and dose-rate dependency of the frequency of HPRT deficient T lymphocytes in the spleen of the ^{137}Cs γ-irradiated mouse. Int J Radiat Biol 66(3):319-326.

Lowe VPW, Horrill AD. 1988. Ecological half-life of caesium in Roe deer (Capreolus capreolus). Environ Pollut 54:81-87.

Luetzelschwab JW. 1976. A field sampling unit that uses an ion-exchange resin to concentrate radioactive ions in river water. Health Phys 31:387-389.

Lundgren DL, Hahn FF, Griffith WC, et al. 1996. Pulmonary carcinogenicity of relatively low doses of beta-particle radiation from inhaled $^{144}CeO_2$ in rats. Radiat Res 146:525-535.

Lundgren DL, Hahn FF, McClellan RO. 1980. Influence of age at the time of inhalation exposure to aerosols of $^{144}CeO_2$ on ^{144}Ce retention, dosimetry and toxicity in mice. Health Phys 38:643-655.

Lymberis C, Makrigiorgos G, Sbonias E, et al. 1987. Radiocesium levels in human muscle samples in Greece one year after the Chernobyl accident. Appl Radiat Isot 39(2):175-176.

*Maenhaut W, Zoller WH, Duce RA, et al. 1979. Concentration and size distribution of particulate trace elements in the south polar atmosphere. J Geophys Res 84(C5):2421-2431.

*Mahara Y. 1993. Heavy metals in the environment: Storage and migration of fallout strontium-90 and cesium-137 for over 40 years in the surface soil of Nagasaki. J Environ Qual 22:722-730.

*Mahlum DD, Sikov MR. 1969. Comparative metabolism of ^{137}Cs by adult, suckling and prenatal rats. Comp Biochem Physiol 30:169-175.

Manion PJ, Hanson LH, Fodale MF. 1988. Sterilizing effects of cesium-137 irradiation on male sea lampreys released in the Big Garlic River Michigan USA. Great Lakes Fish Comm Tech Bull 53:1-8.

Marouf BA, Mohamad AS, Taha JS, et al. 1992. The transfer of ^{137}Cs from soil to plants. Environ Int 18:211-215.

Marshall GC, Kessler WV, Born GS, et al. 1997. Effects of altered thyroid states on cesium-137 accumulation in the liver of rats. Health Phys 33:622-624.

Marshall H, Cooper MB. 1989. Cs^{137} in the barren ground caribou of Canada. Health Phys 56(Suppl. 1):S67-S68.

*Matsuda Y, Tobari I, Yamagiwa J, et al. 1985. Dose-response relationship of γ-ray-induced reciprocal translocations at low doses in spermatogonia of the crab-eating monkey (*Macaca fascicularis*). Mutat Res 151:121-127.

Matsumoto K, Yagi M. 1991. Instrumental photon activation analysis of coal fly ashes using the internal standard method coupled with the standard addition method. J Radioanal Nucl Chem 152(1):55-66.

9. REFERENCES

Matsunaga T, Ueno T, Chandradjith RLR, et al. 1999. Cesium-137 and mercury contamination in lake sediments. Chemosphere 39(2):269-283.

Matsusaka N, Inaba J. 1967. Whole-body retention of caesium-137 in suckling and weanling mice. Nature 214:303-304.

Matsusaka N, Yamakawa Y, Sato I, et al. 1997. Organ distribution of ^{137}Cs in mouse fetuses and their dams. Radioisotopes 46:214-218.

Mayes RW, Eayres HF, Beresford NA, et al. 1992. Changes with age in the absorption of radiocesium by sheep. Radiat Prot Dosim 41:83-86.

*Mayr U, Butsch A, Schneider S. 1992. Validation of two in vitro test systems for estrogenic activities with zearalenone, phytoestrogens and cereal extracts. Toxicology 74:135-149.

McAuly IR, Doyle C. 1985. Radiocesium levels in Irish sea fish and the resulting dose to the population of the Irish Republic. Health Phys 48(3):333-337.

McCartney M, Kershaw PJ, Woodhead DS, et al. 1994. Artificial radionuclides in the surface sediments of the Irish Sea, 1968-1988. Sci Total Environ 141:103-138.

McClellan RO, Rupprecht FC. 1968. Toxicity of ^{137}CsCl in the beagle dog. In: Fission Product Inhalation Program, ed. Fission product inhalation program annual report 1967-1968. Albuquerque, NM: Lovelace Foundation for Medical Research and Education, 54-75.

*McCraw TF. 1965. The half-time of cesium-137 in man. Radiol Health Data Rep 6(12):711-718.

*McCreedy CD, Jagoe CH, Glickman LT, et al. 1997. Bioaccumulation of cesium137 in yellow bullhead catfish (Ameiurus natalis) inhabiting an abandoned nuclear reactor reservoir. Environ Toxicol Chem 16(2):328-335.

*McGee EJ, Synnott HJ, Keatinge M, et al. 1993. Persistence and prediction of radiocaesium levels in animals grazing semi-natural environments. Sci Total Environ 138:91-99.

McGee EJ, Synnott HJ, O'Keefe C, et al. 1995. Radionuclide uptake by red deer (Cervus elaphus) on mountain grazing. Br Vet J 151:671-682.

Meiler CF, Katzung BG. 1981. Cesium blockade of delayed outward currents and electrically induced pacemaker activity in mammalian ventricular myocardium. J Gen Physiol 77:531-547.

Mejstrik V, Svacha J. 1988. Concentrations of ^{232}Th, ^{226}Ra, ^{137}Cs and ^{40}K in soils, and radioactivity in areas of coal-fired power plants. Sci Total Environ 72:69-79.

Melin J, Wallberg L, Suomela J. 1994. Distribution and retention of cesium and strontium in Swedish boreal forest ecosystems. Sci Total Environ 157:93-105.

*Melo DR, Lipsztein JL, Oliveira CAN, et al. 1994. A ^{137}Cs age-dependent biokinetic study. Health Phys 66(6):S25-S26.

Melo DR, Lipsztein JL, Oliveira CAN, et al. 1995. ^{137}Cs metabolism in pregnant women. Health Phys 68(6):S77.

9. REFERENCES

*Melo DR, Lipsztein JL, Oliveira CAN, et al. 1997. A biokinetic model for ^{137}Cs. Health Phys 73(2):320-332.

*Melo DR, Lundgren DL, Muggenburg BA, et al. 1996. Prussian blue decorporation of ^{137}Cs in beagles of different ages. Health Phys 71(2):190-197.

Messiha FS. 1976. Distribution and retention of exogenously administered alkali metal ions in the mouse brain. Arch Int Pharmacodyn 219:87-96.

*Messiha FS. 1978. Anti-depressant action of caesium chloride and its modification of chlorpromazine toxicity in mice. Br J Pharmacol 64:9-12.

Messiha FS. 1985. Cesium: An overview. Neurol Neurobiol 15:247-257.

Messiha FS. 1988a. Effect of separate and combined maternal ingestion of alkali metals on the developing mouse. Neurosci Biobehav Rev 12:205-208.

*Messiha FS. 1988b. Maternal cesium chloride ingestion and the newborn. Neurosci Biobehav Rev 12:209-213.

Messiha FS. 1989a. Maternally-mediated neonatal lithium-cesium interaction in the mouse. Physiol Behav 46:89-95.

*Messiha FS. 1989b. A toxicology evaluation of postnatal maternal exposure to cesium. Physiol Behav 46:85-88.

Messiha FS. 1994. Developmental toxicity of cesium in the mouse. Gen Pharmac 25(3):395-400.

Messiha FS, Larson JW. 1976. A pharmacokinetic study of rubidium and cesium salts. Proc West Pharmacol Soc 19:108-112.

*MI Dept Environ Quality. 2000. Maximum contaminant levels; incorporation by reference. Safe drinking water act. State of Michigan Department of Environmental Quality. Http://www.deq.state.mi.us/. June 14, 2000.

Mietelski JW, Jasinska M, Kubica B, et al. 1994. Radioactive contamination of Polish mushrooms. Sci Total Environ 157:217-226.

*Mihok S, Schwartz B, Wiewel AM. 1989. Bioconcentration of fallout ^{137}Cs by fungi and red-backed voles (clethrionomys gapperi). Health Phys 57(6):959-966.

*Mikamo K, Kamiguchi Y, Tateno H. 1990. Spontaneous and in vitro radiation-induced chromosome aberrations in human spermatozoa: Application of a new method. In: Mendelsohn ML, Albertini RJ, eds. Mutation and the environment: Part B: Metabolism, testing methods, and chromosomes. New York, NY: Wiley-Liss, 447-456.

*Mikamo K, Kamiguchi Y, Tateno H. 1991. The interspecific in vitro fertilization system to measure human sperm chromosomal damage. In: Gledhill BL, Mauro F, ed. New horizons in biological dosimetry. New York, NY: Wiley-Liss, 531-542.

9. REFERENCES

*Mikhaylovskaya LN, Molchanova IV, Karavayeva YN. 1993. Forms of occurrence and migration of radionuclides in soils affected by the accident at the Chernobyl nuclear power plant. Euras Soil Sci 25(7):103-107.

Millard GC, Fraley L, Markham OD. 1983. Deposition and retention of ^{141}Ce and ^{134}Cs aerosols on cool desert vegetation. Health Phys 44(4):349-357.

*Miller CE. 1964. Retention and distribution of ^{137}Cs after accidental inhalation. Health Phys 10:1065-1070.

Miltenberger RP, Lessard ET, Greenhouse NA. 1981. ^{60}Co and ^{137}Cs long-term biological removal rate constants for the United Nations scientific committee on the Marshallese population. Health Phys 40:615-623.

*Minamisawa T, Hirokaga K, Sasaki S. 1990. Gross morphological changes of the mouse brain exposed prenatally to ionizing radiation. J Radiat Res 31:214-218.

*Minamisawa T, Hirokaga K, Sasaki S, et al. 1992. Effects of fetal exposure to gamma rays on aggressive behavior in adult male mice. J Radiat Res 33:243-249.

*Molero J, Sanchez-Cabeza JA, Merino J, et al. 1999. Impact of ^{134}Cs and ^{137}Cs from the Chernobyl reactor accident on the Spanish Mediterranean marine environment. J Environ Radioact 43:357-370.

Monakhov AS, Iakovleva TK, Strekalov SA, et al. 1991. The cytogenetic and hematotoxic effect of cesium137 incorporated in the rat body. Radiobiologiia 31(6):798-802.

*Mondon KJ, Walters B. 1990. Measurement of radiocaesium, radiostrontium, and plutonium in whole diets, following deposition of radioactivity in the UK originating from the Chernobyl power plant accident. Food Addit Contam 7(6):837-848.

*Moore W, Comar CL. 1962. Absorption of caesium 137 from the gastro-intestinal tract of the rat. Int J Radiat Biol 5(3):247-254.

*Moore W, Comar CL. 1963. Movement of ^{137}Cs across surviving intestinal segments *in vitro*. Int J Radiat Biol 6(6):507-511.

Morgan IJ, Tytler P, Bell MV. 1993. The accumulation of 137-caesium from fresh water by alevins and fry of Atlantic salmon and brown trout. J Fish Biol 43:877-888.

*Morselli PL, Franco-Morselli R, Bossi L. 1980. Clinical pharmacokinetics in newborns and infants: Age-related differences and therapeutic implications. Clin Pharmacokin 5:485-527.

Motelica-Heino M, Le Coustumer P, Thomassin JH, et al. 1998. Macro and microchemistry of trace metals in vitrified domestic wastes by laser ablation ICP-MS and scanning electron microprobe x-ray energy dispersive spectroscopy. Talanta 46:407-422.

Muggenburg BA, Boecker BB, Hahn FF, et al. 1983. The risk of liver tumors in dogs and man from radioactive aerosols. In: Thompson RC, Mahaffey JA, eds. Life-span radiation effects studies in animals: What can they tell us? Proceedings of the twenty-second Hanford life science symposium held at Richland, Washington, September 27-29, 1983. Hanford Life Sciences Symposium 22nd. Springfield, VA: US Department of Energy, 556-563.

9. REFERENCES

*Mumma RO, Raupach DC, Sahadewan K, et al. 1990. National survey of elements and radioactivity in municipal incinerator ashes. Arch Environ Contam Toxicol 19:399-404.

*Mumma RO, Raupach DC, Sahadewan K, et al. 1991. Variation in elemental composition of municipal refuse incinerator ashes with time of sampling. Chemosphere 23(3):391-395.

*Murakawa Y, Sezaki K, Yamashita T, et al. 1997. Three-dimensional activation sequence of cesium-induced ventricular arrhythmias. Am J Physiol 42:H1377-H1385.

Muramatsu Y, Yoshida S, Sumiya M. 1991. Concentrations of radiocesium and potassium in basidiomycetes collected in Japan. Sci Total Environ 105:29-39.

Myers B, Shaw SM. 1975. Deposition, clearance, and distribution of cesium-137 labeled cesium chloroplatinate in normal and sulfur dioxide exposed rats. AIChE Symp Ser 71(147):107-110.

Nakaoka A, Yokoyama H, Fukushima M, et al. 1980. Rapid determination method of radiocesium in sea water by cesium-selective resin. J Radioanal Chem 56(1-2):13-24.

*NAS/NRC. 1989. Report of the oversight committee. In: Biologic markers in reproductive toxicology. Washington, DC: National Academy Press. National Academy of Sciences. National Research Council.

*Natarajan AT, Santos SJ, Darroudi F, et al. 1998. Cesium-induced chromosome aberrations analyzed by flourescence in situ hybridization: Eight years follow up of the Goiania radiation accident victims. Mutat Res 400:299-312.

Nayebpour M, Solymoss BC, Nattel S. 1989. Cardiovascular and metabolic effects of caesium chloride injection in dogs - limitations as a model for the long QT syndrome. Cardiovasc Res 23:756-766.

*NCRP. 1977. Cesium-137 from the environment to man: Metabolism and dose. Bethesda, MD: National Council on Radiation Protection and Measurements. NCRP Report No. 52.

*NCRP. 1985. General concepts for the dosimetry of internally deposited radionuclides. Bethesda, MD: National Council on Radiation Protection and Measurements. NCRP Report No. 84.

*NCRP. 1987. Use of bioassay procedures for assessment of internal radionuclide deposition. Bethesda, MD: National Council on Radiation Protection and Measurements. NCRP Report No. 87.

*NCRP. 1997. Deposition, retention and dosimetry of inhaled radioactive substances. Bathesda, MD: National Council on Radiation Protection and Measurements. NCRP Report No. 125.

Nellessen JE, Fletcher JS. 1993. Assessment of published literature on the uptake, accumulation, and translocation of heavy metals by vascular plants. Chemosphere 27(9):1669-1680.

Nelson A, Ullberg S, Kristoffersson H, et al. 1961. Distribution of radiocesium in mice. Acta Radiol 55:374-384.

*Neulieb R. 1984. Effects of oral intake of cesium chloride: A single case report. Pharmacol Biochem Behav 21(Suppl. 1):15-16.

*Nevissi AE. 1992. Measurement of actinides and long-lived radionuclides in large coral samples. J Radioanal Nucl Chem 156(2):243-251.

Newman G. 1996. Increased morbidity following the introduction of remote afterloading, with increased dose rate, for cancer of the cervix. Radiother Oncol 39:97-103.

Ng YC, Anspaugh LR, Cederwall RT. 1990. ORERP internal dose estimates for individuals. Health Phys 59(5):693-713.

Nielson SP, Gryning SE, Karlberg O, et al. 1990. Dry deposition of ^{88}Rb and ^{138}Cs from a boiling water reactor plume. Health Phys 58(3):283-289.

Nifontova M. 1995. Radionuclides in the moss-lichen cover of tundra communities in the Yamal Peninsula. Sci Total Environ 160/161:749-752.

Niimi AJ. 1987. Biological half-lives of chemicals in fishes. Rev Environ Contam Toxicol 99:1-46.

*Nikula KJ, Muggenburg BA, Chang I-Y, et al. 1995. Biological effects of ^{137}CsCl injected in beagle dogs. Radiat Res 142:347-361.

*Nikula KJ, Muggenburg BA, Griffith WC, et al. 1996. Biological effects of ^{137}CsCl injected in beagle dogs of different ages. Radiat Res 146:536-547.

Nimis PL, Bolognini G, Giovani C. 1994. Radiocontamination patterns of vascular plants in a forest ecosystem. Sci Total Environ 157:181-188.

*NIOSH. 2000. Cesium. NIOSH pocket guide to chemical hazards. National Institute for Occupational Safety and Health. Http://www.cdc.gov/niosh/npg/npgd0145.html. October 13, 2000.

*NOES. 1989. National Occupational Exposure Survey. National Institute for Occupational Safety and Health.

*Norton S, Kimler BF. 1987. Correlation of behavior with brain damage after in utero exposure to toxic agents. Neurotoxicol Teratol 9:145-150.

*Norton S, Kimler BF. 1988. Comparison of functional and morphological deficits in the rat after gestational exposure to ionizing radiation. Neurotoxicol Teratol 10:363-371.

Norton S, Kimler BF. 1990. Early effects of low doses of ionizing radiation on the fetal cerebral cortex in rats. Radiat Res 124:235-241.

*NRC. 1983. Metabolic data and retention functions for the intracellular alkali metals. Washington, DC: U.S. Nuclear Regulatory Commission. NUREG/CR-3245. ORNL/TM-8630. DOE 40-550-75.

*NRC. 1984. Lower limit of detection: Definition and elaboration of a proposed position for radiological effluent and environmental measurements. Washington, DC: U.S. Nuclear Regulatory Commission. NUREG/CR-4604.

*NRC. 1991. Nuclear regulatory commission digest. U.S. Nuclear Regulatory Commission. Code of Federal Regulations. 10 CFR 20, appendix B.

*NRC. 1993a. Pesticides in the diets of infants and children. Washington, DC: National Academy Press. National Research Council.

9. REFERENCES

*NRC. 1993b. Radioactive materials released from nuclear power plants: Annual report 1993. Washington, DC: U.S. Nuclear Regulatory Commission. NUREG/CR-2907. BNL-NUREG-51581.

*NRC. 1997. Minimum detectable concentrations with typical radiation survey instruments for various contaminants and field conditions. Rockville, MD: Nuclear Regulatory Commission. U.S. Report NUREG-1507.

*NRC. 1998. Probabilistic accident consequence uncertainty analysis: Uncertainty assessment for internal dosimetry. Vol 1 and 2. Commission of European Communities. Nuclear Regulatory Committee. NUREG/CR-6571. EUR 16773. SAND98-0119.

*NRC. 1999a. Annual limits on intake (ALIs) and derived air concentrations (DACs) of radionuclides for occupational exposure; Effluent concentrations; Concentrations for release to sewerage. Nuclear Regulatory Commission. Code of Federal Regulations. 10 CFR 20 App B.

*NRC. 1999b. Quantities of licensed material requiring labeling. Nuclear Regulatory Commission. Code of Federal Regulations. 10 CFR 20 App C.

Nugis VY, Konchalovskii MV. 1993. Biological dosimetry, diagnosis, and treatment of bone-marrow syndrome in victims of the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 349-384.

O'Brien DJ, Kaneene JB, Poppenga RH. 1993. The use of mammals as sentinels for human exposure to toxic contaminants in the environment. Environ Health Perspect 99:351-368.

OECD. 1995. Chernobyl ten years on, radiological and health impact. Paris: Organization for Economical Cooperation and Development.

Ohno T, Hess CT. 1994. Levels of ^{137}Cs and ^{40}K in wood ash-amended soils. Sci Total Environ 152:119-123.

*Olivier P, Marzin D. 1987. Study of the genotoxic potential of 48 inorganic derivatives with the SOS chromotest. Mutat Res 189:263-269.

*Olsen CR, Thein M, Larsen IL, et al. 1989. Plutonium, lead-210, and carbon isotopes in the Savannah Estuary: Riverborne versus marine sources. Environ Sci Technol 23:1475-1481.

*Ondov JM, Choquette CE, Zoller WH, et al. 1989. Atmospheric behavior of trace elements on particles emitted from a coal-fired power plant. Atmos Environ 23(10):2193-2204.

Oostdam JV, Gilman A, Dewailly E, et al. 1999. Human health implications of environmental contaminants in Arctic Canada: A review. Sci Total Environ 230:1-82.

Orel VE, Tereschenki VM, Czyatkovskaya NN, et al. 1998. Physical dosimetry and biological indicators of carcinogenic risk in a cohort of persons exposed to unhealthy ecological factors following the Chernobyl nuclear power plant accident. Arch Environ Health 53(6):398-404.

Osanov DP, Kyruchkov VP, Panova VP. 1990. Skin absorbed doses from ground-deposited radioactive contaminants. At Energ 68(2):107-111.

9. REFERENCES

Osanov DP, Krjuchkov VP, Shaks AI. 1993. Determination of beta radiation doses received by personnel involved in the mitigation of the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 313-346.

*OTA. 1990. Neurotoxicity: Identifying and controlling poisons of the nervous system. Washington, DC: Office of Technology Assessment. OTA-BA-438.

*Oughton DH, Day JP. 1993. Determination of cesium, rubidium and scandium in biological and environmental materials by neutron activation analysis. J Radioanal Nucl Chem 174(1):177-185.

*Owen GM, Brozek J. 1966. Influence of age, sex and nutrition on body composition during childhood and adolescence. In: Falkner F, ed. Human development. Philadelphia, PA: WB Saunders, 222-238.

*Owens PN, Walling DE, He Q. 1996. The behaviour of bomb-derived caesium-137 fallout in catchment soils. J Environ Radioact 32(3):169-191.

Paasikallio A. 1984. The effect of time on the availability of ^{90}Sr and ^{137}Cs to plants from Finnish soils. Ann Agric Fenn 23(2):109-120.

*Paasikallio A. 1999. Effect of biotite, zeolite, heavy clay, bentonite and apatite on the uptake of radiocesium by grass from peat soil. Plant Soil 206:213-222.

*Paasikallio A, Rantavaara A, Sippola J. 1994. The transfer of cesium-137 and strontium-90 from soil to food crops after the Chernobyl accident. Sci Total Environ 155:109-124.

Pacyna JM. 1995. The origin of Arctic air pollutants: lessons learned and future research. Sci Total Environ 160/161:39-53.

*Padovani L, Caprossi D, Tedeschi B, et al. 1993. Cytogenetic study in lymphocytes from children exposed to ionizing radiation after the Chernobyl accident. Mutat Res 319:55-60.

*Palmer HE, Branson BM, Cohn SH, et al. 1976. Standard field methods for determining ^{137}Cs and ^{131}I *in vivo*. Health Phys 30:113.

*Papastefanou C, Manolopoulou M, Ioannidou A, et al. 1989. Time-dependent radioactive concentrations of fallout following the Chernobyl reactor accident. Sci Total Environ 84:283-289.

*Papastefanou C, Manolopoulou M, Sawidis T. 1992. Residence time and uptake of ^{137}Cs in lichens and mosses at temperate latitude (40°N). Environ Int 18:397-401.

Paschal DC, Ting BG, Morrow JC, et al. 1996. Trace metals in urine of United States residents: Reference range concentrations. Environ Res A76:53-59.

*Patterson E, Szabo B, Scherlag BJ, et al. 1990. Early and delayed after depolarizations associated with cesium chloride-induced arrhythmias in the dog. J Cardiovasc Pharmacol 15:323-331.

Paunow S, Laussmann D. 1980. Chromosomal bone-marrow changes after ^{137}Cs incorporation in dsungaric hamsters. Radiobiol Radiother 21(1):30-35.

*Pearce J. 1995. Radiocesium in migratory bird species in northern Ireland following the Chernobyl accident. Bull Environ Contam Toxicol 54:805-811.

*Pendic B, Milivojevic K. 1966. Contamination interne au ^{137}Cs par voie transcutanée et effet des moyens de décontamination et de protection sur la résorption transcutanée de ce radionuclide. Health Phys 12:1829-1830.

Pennington JAT, Capar SG, Parfitt CH, et al. 1996. History of the Food and Drug Administration's total diet study (part II), 1987-1993. J AOAC Int 79(1):163-170.

*Penttila S, Kairesalo T, Uusi-Rauva A. 1993. The occurrence and bioavailability of radioactive ^{137}Cs in small forest lakes in southern Finland. Environ Pollut 82:47-55.

*Petrow HG, Levine H. 1967. Ammonium hexacyanocobalt ferrate as an improved inorganic exchange material for determination of cesium-137. Anal Chem 39:360-362.

Pham MK, Garnier J-M. 1998. Distribution of trace elements associated with dissolved compounds (<0.45µm-1nm) in freshwater using coupled (frontal cascade) ultrafiltration and chromatographic separations. Environ Sci Technol 32(4):440-449.

*Pietrzak-Flis Z, Krajewski P, Krajewska G, et al. 1994. Transfer of radiocesium from uncultivated soils to grass after the Chernobyl accident. Sci Total Environ 141:147-153.

Pietrzak-Flis Z, Rostek J, Lada W. 1988. Estimation of ^{137}Cs and ^{134}Cs body burden in Warsaw after the Chernobyl accident. Radiat Prot Dosim 25:101-105.

Pinsky C, Bose R. 1984. Pharmacological and toxicological investigations of cesium. Pharmacol Biochem Behav 21(Suppl. 1):17-23.

*Pinsky C, Bose R, Dua AK, et al. 1980. Interdisciplinary studies on brain level determination and behavioral effects of cesium (Cs). Pharmacologist 22(3):158.

Pinsky C, Bose R, Taylor JR, et al. 1981. Cesium in mammals: Acute toxicity, organ changes and tissue accumulation. J Environ Sci Health Part A 16(5):549-567.

Pitkyanen GB. 1978. Effect of chronic irradiation of a pike *Exxox lucius L.* on its reproductive function. Tr Inst Ekol Rast Zhivotn, Ural Nauchn Tsentr, Akad Nauk SSSR 114:74-78.

*Platford RF, Joshi SR. 1989. Radionuclide partitioning across Great Lakes natural interfaces. Environ Geol Water Sci 14(3):183-186.

Pohl-Ruling J, Haas O, Brogger A, et al. 1991. The effect on lymphocyte chromosomes of additional radiation burden due to fallout in Salzburg (Austria) from the Chernobyl accident. Mutat Res 262:209-217.

Polikarpov GG, Tsytsugina VG. 1993. Distribution of chromosome aberrations among hydrobiont cells under the effect of ionizing radiation and chemical mutagens of the environment. Radiobiologiia 33(2):205-213.

Polyák K, Bódog I, Hlavay J. 1994. Determination of chemical species of selected trace elements in fly ash. Talanta 41(7):1151-1159.

9. REFERENCES

Pomerantseva MD, Ramaiya LK, Chekhovich AV, et al. 1993. Genetic effect of incorporated ^{137}Cs in male mice after single injection of the isotope. Radiat Biol Radioecol 33(1):567-576.

Popov VY, Kutnyakov IV, Zhirnov VG, et al. 1995. Vertical distribution of ^{90}Sr and ^{137}Cs in alluvial sod soils in the near zone of the Chernobyl nuclear power plant. Euras Soil Sci 27(11):65-73.

Poston TM, Klopfer DC. 1988. Concentration factors used in the assessment of radiation dose to consumers of fish: A review of 27 radionuclides. Health Phys 55(5):751-766.

Preston RJ, Dean BJ, Galloway S, et al. 1987. Mammalian in vivo cytogenetic assays: Analysis of chromosome aberrations in bone marrow cells. Mutat Res 189:157-165.

Prisjazhniuk AY. 1993. Spatio-temporal models for incidence of malignant neoplasms in the area subjected to radioactive contamination after the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 399-423.

*Prister BS, Omel'Yanenko NP, Yatnikova P. 1990. Migration of radionuclides in soil and their transfer to plants within the zone of the Chernobyl nuclear power plant accident. Sov Soil Sci 23(2):76-86.

*Probst T, Zeh P, Kim J-I. 1995. Multielemental determinations in ground water ultrafiltrates using inductively coupled plasma mass spectrometry and monostandard neutron activation analysis. Fresenius J Anal Chem 351:745-751.

Quastel MR, Goldsmith JR, Mirkin L, et al. 1997. Thyroid-stimulating hormone levels in children from Chernobyl. Environ Health Perspect Suppl 6:1497-1498.

*Rabitsch H, Feenstra O, Kahr G. 1991. Radiocesium levels in humans over a four-year period. J Nucl Med 32:1491-1495.

*Rabon EW. 1978. Calcium, strontium-89, strontium-90, and cesium-137 in pregnant white-tailed dear and related fetuses. DOE Symp Ser 45:682-690.

*Rabon EW, Johnson JE. 1973. Rapid field-monitoring of cesium-137 in white-tailed deer. Health Phys 25:515-516.

Raghavendran KV, Satbhai PD, Abhyankar B, et al. 1978. Long-term retention studies of ^{131}I and ^{137}Cs and ^{60}Co in Indian workers. Health Phys 34:185-188.

*Rahola T, Suomela M. 1998. The ^{137}Cs content in Finnish people consuming foodstuffs of wild origin. Radiat Prot Dosim 79:187-189.

Ramaiya LK, Pomerantseva MD, Chekhovich AV, et al. 1993. [Genetic efficiency of incorporated ^{137}Cs in male mice after long-term administration of the isotope.] Radiat Biol Radioecol 33(1):577-584. (Russian).

*Ramaiya LK, Pomerantseva MD, Chekhovich AV, et al. 1994. Genetic effects of testicular incorporation of ^{137}Cs in mice. Mutat Res 324:139-145.

Ramalho AT, Nascimento ACH. 1991. The fate of chromosomal aberrations in ^{137}Cs-exposed individuals in the Goliania radiation accident. Health Phys 60(1):67-70.

9. REFERENCES

Ramalho AT, Nascimento ACH, Littlefield LG, et al. 1991. Frequency of chromosomal aberrations in a subject accidentally exposed to ^{137}Cs in the Goiania (Brazil) radiation accident: Intercomparison among four laboratories. Mutat Res 252:157-160.

Ramalho AT, Nascimento ACH, Natarajan AT. 1988. Dose assessments by cytogenic analysis in the Goiania (Brazil) radiation accident. Radiat Prot Dosim 25:97-100.

Ramboiu S, Derevenco P, Bordas E, et al. 1990. Effects of ingestion of radioactive cesium on behavioral indices in rats. Rev Roum Physiol 27(1):29-37.

Ramzaev PV, Tarasov SI. 1976. [Hygienic evaluation of the combined effect of different types of harmful factors]. In: Liberman AN, ed. [Hygienic evaluation of factors of radiation and nonradiation natures and their combinations]. Leningrad, USSR: Leningradskii Nauchno-Issledovatel'skii Institut Radiatsionnoi Gigieny, 5-10. (Russian)

*Rank D, Maringer FJ, Terlunen J. 1990. The radioactivity of sediments in Danube reservoirs in Austria before and after the Chernobyl accident. Water Sci Technol 22(5):211-218.

*Rastogi RB, Singhal RL, Lapierre YD. 1980. Effects of rubidium and cesium on central catecholamines and locomotor behavior in rats. J Neurochem 34(6):1764-1767.

Raven KP, Loeppert RH. 1997. Trace element composition of fertilizers and soil amendments. J Environ Qual 26:551-557.

Reddi PR, Nussbaum H, Wollin M, et al. 1974. Treatment of carcinoma of the cervix uteri with special reference to radium system. Obstet Gynecol 43(2):238-247.

*Redman HC, McClellan RO, Jones RK, et al. 1972. Toxicity of ^{137}CsCl in the beagle. Early biological effects. Radiat Res 50:620-648.

Reish DJ, Geesey GG, Wilkes FG, et al. 1982. Marine and estuarine pollution. J Water Pollut Control Fed 54(6):786-811.

Remez VP, Sapozhnikov YA. 1996. The rapid determination of caesium radionuclides in water systems using composite sorbents. Appl Radiat Isot 47(9-10):885-886.

*Richmond CR, Furchner JE. 1961. Enhancement of cesium^{137} excretion by rats fed potassium-supplemented diets. Proc Soc Exp Biol Med 108:797-798.

*Richmond CR, Furchner JE, Langham WH. 1962. Long-term retention of radiocesium by man. Health Phys 8:201-205.

*Richter T, Schroder G, Kaminski S, et al. 1993. Transport of particles contaminated with cesium radionuclides into the sediment of Lake Constance. Water Sci Technol 28:117-121.

*Riga-Karandinos AN, Karandinos MG. 1998. Caesium-137 concentrations and ecological half-lives in three epiphytic lichen species from southern Greece (Megalopolis). Bryologist 101(3):422-427.

Riget F, Johansen P, Asmund G. 1997. Baseline levels and natural variability of elements in three seaweed species from west Greenland. Mar Pollut Bull 34(3):171-176.

9. REFERENCES

*Risica S, Grisanti G, Tancredi F, et al. 1994. A study on some stable elements and radionuclides in human milk. ACH Models Chem 131:651-660.

Rivera J. 1965. Dietary intakes and body burdens of cesium-137. Radiol Health Data Rep 6(9):504-506.

*Robb P, Owen LMW, Crews HM. 1995. Stable isotope approach to fission product element studies of soil-to-plant transfer and *in vitro* modelling of ruminant digestion using inductively coupled plasma mass spectrometry. J Anal Atom Spectrom 10:625-629.

*Robbins JA, Mudroch A, Oliver BG. 1990. Transport and storage of ^{137}Cs and ^{210}Pb in sediments of Lake St. Clair. Can J Fish Aquat Sci 47:572-587.

Roberto B, Gemignani F, Morizzo C, et al. 1998. Cytogenetic damage in lymphocytes of healthy and thyroid tumor-affected children from the Gomel region (Belarus). Mutat Res 405:89-95.

*Robinson WL, Phillips WA. 1989. Estimates of the radiological dose form ingestion of ^{137}Cs and ^{90}Sr to infants, children, and adults in the Marshall Islands. Livermore, CA: Lawrence Livermore National Laboratory.

Roca MC, Vallejo VR, Roig M, et al. 1997. Prediction of cesium-134 and strontium-85 crop uptake based on soil properties. J Environ Qual 26:1354-1362.

Romanenko AM, Gaiyrov AG. 1998. Particular features of morphogenesis of transition cell tumors of human urinary bladder in Ukraine after Chernobyl accident. Lik Sprava 26(3):11-14.

*Ropolo R, Cesana P. 1997. ^{137}Cs urinary excretion by northwestern Italians ten years after the Chernobyl accident. Health Phys 73(3):498-501.

Rosen K, Eriksson A, Haak E. 1996. Transfer of radiocesium in sensitive agricultural environments after the Chernobyl fallout in Sweden. I. County of Gavleborg. Sci Total Environ 182:117-133.

Rosen K, Haak E, Eriksson A. 1998. Transfer of radiocesium in sensitive agricultural environments after the Chernobyl fallout in Sweden: III. County of Vasternorrland. Sci Total Environ 209:91-105.

*Rosenthal JJ, de Almeida CE, Mendonca AH. 1991. The radiological accident in Goiania: The initial remedial actions. Health Phys 60(1):7-15.

*Rosoff B, Cohn SH, Spencer H. 1963. Cesium-137 in man. Radiat Res 19:643-654.

*Rowan DJ, Chant LA, Rasmussen JB. 1998. The fate of radiocesium in freshwater communities- why is biomagnification variable both within and between species? J Environ Radioact 40(1):15-36.

Rowan JS, Walling DE. 1992. The transport and fluvial redistribution of Chernobyl-derived radiocesium within the River Wye basin, UK. Sci Total Environ 121:109-131.

Ruhm W, Kammerer L, Hiersch L, et al. 1996. Migration of ^{137}Cs and ^{134}Cs in different forest soil layers. J Environ Radioact 33(1):63-75.

*Rundo J. 1964. A survey of the metabolism of caesium in man. Br J Radiol 37:108-114.

Rundo J. 1965. A case of accidental inhalation of irradiated uranium. Br J Radiol 38:39-50.

9. REFERENCES

*Rundo J, Newton D. 1964. Increase in the cesium-137 content of man due to radioactive fallout, 1962-1964. Nature 203:537-538.

Rundo J, Richmond CR. 1970. Altitude effect on the biological half life of caesium in man. Nature 225:83-84.

*Rundo J, Turner FM. 1966. Metabolism of caesium-137 during pregnancy. British Atomic Energy Research Establishment Progress Report, AEKE-PR/HPM 11, 32(1966).

Rundo J, Turner FM. 1967. Metabolism of caesium-137 during pregnancy. British Atomic Energy Research Establishment Progress Report, AEKE-PR/HPM 12, 34(1967).

*Rundo J, Mason JI, Newton D, et al. 1963. Biological half-life of caesium in man in acute chronic exposure. Nature 200:188-189.

*Ruse ME, Peart MR. 2000. Intrasite sampling of Hong Kong soils contaminated by caesium-137. Chemosphere 41:45-51.

Russell WL, Kelly EM. 1982. Mutation frequencies in male mice and the estimation of genetic hazards of radiation in men. Proc Natl Acad Sci U S A 79:542-544.

*Rybacek K, Wilhelmova L, Tomasek M. 1994. Behavior of radiocesium in atmospheric aerosol and fallout in Prague. J Radioanal Nucl Chem 186(3):245-255.

*Saad AY, Abdelazim AA, El-Khashab MM, et al. 1991. Effects of gamma radiation on incisor development of the prenatal albino mouse. J Oral Pathol Med 20:385-388.

*Saad AY, Abdelazim AA, El-Khashab MM, et al. 1994. Induction of cleft palate by gamma-irradiation of prenatal CD-1 mice. Cleft Palate Craniofac J 31(5):351-355.

Sachdev P, Sachdev MS, Deb DL. 1995. Adsorption-desorption of radiocaesium (^{137}Cs) in semi-arid and tropical soils. J Nucl Agric Biol 24(4):201-209.

Sachdev P, Sachdev MS, Deb DL. 1998. The uptake and transfer of caesium-137, strontium-90 and zinc-65 from soil to food crops in tropical environment. J Nucl Agric Biol 27(1):1-9.

Sanchez AL, Schell WR. 1986. Uptake mechanisms for Co-60, Sr-85, Cs-137 on peat. Health Phys 50(Suppl. 1):S87.

*Sanchez AL, Wright SM, Smolders E, et al. 1999. High plant uptake of radiocesium from organic soils due to Cs mobility and low soil K content. Environ Sci Technol 33:2752-2757.

*Sarkka J, Keskitalo A, Luukko A. 1996. Temporal changes in concentration of radiocesium in lake sediment and fish of southern Finland as related to environmental factors. Sci Total Environ 191:125-136.

Sauras T, Roca MC, Tent J, et al. 1994. Migration study of radionuclides in a Mediterranean forest soil using synthetic aerosols. Sci Total Environ 157:231-238.

*Sawidis T, Drossos E, Heinrich G, et al. 1990. Cesium-137 accumulation in higher plants before and after Chernobyl. Environ Int 16:163-169.

9. REFERENCES

Sbrignadello G, Degetto S, Battiston GA, et al. 1994. Distribution of ^{210}Pb and ^{137}Cs in snow and soil samples from Antarctica. Int J Environ Anal Chem 55:235-242.

Schell WR, Tobin MJ, Massey CD. 1989. Evaluation of trace metal deposition history and potential element mobility in selected cores from peat and wetland ecosystems. Sci Total Environ 87/88:19-42.

Schimmack W, Bunzl K. 1992. Migration of radiocesium in two forest soils as obtained from field and column investigations. Sci Total Environ 116:93-107.

*Schull WJ, Otake M. 1999. Cognitive function and prenatal exposure to ionizing radiation. Teratology 59:222-226.

*Schull WJ, Otake M, Yoshimaru H. 1988. Effect on intelligence test score of prenatal exposure to ionizing radiation in Hiroshima and Nagasaki: A comparison of the T65DR and DS86 dosimetry systems. Radiation Effects Research Foundation. RERF TR 3-88. Research project 24-62.

*Schuller P, Ellies A, Kirchner G. 1997. Vertical migration of fallout ^{137}Cs in agricultural soils from Southern Chile. Sci Total Environ 193:197-205.

Schwarz G, Dunning DE. 1982. Imprecision in estimates of dose ingested ^{137}Cs due to variability in human biological characteristics. Health Phys 43(5):631-645.

Seel JF, Whicker FW, Adriano DC. 1995. Uptake of ^{137}Cs in vegetable crops grown on a contaminated lakebed. Health Phys 68(6):793-799.

Seelye JG, Hesselberg RJ, Mac MJ. 1982. Accumulation by fish of contaminants released from dredged sediments. Environ Sci Technol 16:459-464.

Sekimoto H, Nakamura H, Takagi N. 1996. Toxicity of radioactive wastes discharged from nuclear energy center in the future equilibrium state. Ann Nucl Energy 23(8):663-668.

Senekowitsch R, Kriegel H. 1984. Diaplacental transfer and distribution of some radionuclides in fetal organs at different stages of gestation - experimental results. EUR 8067:183-197.

*Setchell BP, Waites GMH. 1975. The blood-testis barrier. In: Creep RO, Astwood EB, Geiger SR, eds. Handbook of physiology: Endocrinology V. Washington, DC: American Physiological Society.

Sevan'Kaev AV, Lloyd DC, Potetnya OI, et al. 1995. Chromosomal aberrations in lymphocytes of residents of areas contaminated by radioactive discharge from the Chernobyl accident. Radiat Prot Dosim 58(4):247-254.

Shah KR, Filby RH, Davis AI. 1971. Determination of trace elements in tea and coffee by neutron activation analysis. Int J Environ Anal Chem 1:63-73.

Shappard SC, Evenden WG, Schwartz WJ. 1995. Ingested soil: Bioavailability of sorbed lead, cadmium, cesium, iodine, and mercury. J Environ Qual 24:498-505.

Sharma A, Das T, Ghosh BB, et al. 1991. Chromosomal alterations induced by newer metals used in industry. Am J Hum Genet 49(4):451.

9. REFERENCES

*Shenber MA, Johanson KJ. 1992. Influence of zeolite on the availability of radiocesium in soil to plants. Sci Total Environ 113:287-295.

Sheppard MI, Thibault DH. 1991. A four-year mobility study of selected trace elements and heavy metals. J Environ Qual 20:101-114.

Sheppard SC, Evenden WG, Schwartz WJ. 1995. Ingested soil: Bioavailability of sorbed lead, cadmium, cesium, iodine, and mercury. J Environ Qual 24:498-505.

Shilov VP. 1980. [Metabolism of ^{137}Cs in pigs in relation to age.] S-kh Biol 15(5):766-769. (Russian).

*Shiraishi K, Tagami K, Ban-nai T, et al. 1997. Daily intakes of ^{134}Cs, ^{137}Cs, ^{40}K, ^{232}Th, and ^{238}U in Ukranian adult males. Health Phys 73(5):814-819.

Shubik VM. 1975. Effect of Cs^{137} on immunological reactivity: (Review of the literature). J Hyg Epidemiol Microbiol Immunol 19(3):271-278.

Shubik VM, Nevstrueva MA, Kal'nitskii SA, et al. 1978a. A comparative study of changes in immunological reactivity during prolonged introduction of radioactive and chemical substances into the organism with drinking water. J Hyg Epidemiol Microbiol Immunol 22(4):408-401.

Shubik VM, Nevstrueva MA, Kal'nitskii SA, et al. 1978b. Comparative study of changes of immunological reactivity during chronic administration of radioactive and chemical substances in the drinking water. Zh Gig Epidemiol Mikrobiol Immunol 22(4):374-380.

Shutov VN, Bruk GY, Balonov MI, et al. 1993. Cesium and strontium radionuclide migration in the agricultural ecosystem and estimation of internal doses to the population. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I, 167-219.

Shutov VN, Bekyasheva TA, Basalayeva LN, et al. 1994. Effect of soil properties on ^{137}Cs and ^{90}Sr uptake by native grasses. Euras Soil Sci 26(7):88-95.

Sikov MR, Meznarich HK, Traub RJ. 1991. Comparison of placental transfer and localization of caesium strontium and iodine in experimental animals and women. Int J Radiat Biol 60(3):553-555.

Simon N, Silverstone SM. 1978. New miniaturized cesium-137 for needle implants. Int J Nucl Med Biol 5:167-169.

Simon SL, Graham JC. 1994. Results of a nationwide radiological monitoring program in the Marshall Islands. Health Phys 66(6):S14.

*Simon SL, Graham JC. 1998. A comparison of aerial and ground level measurements of ^{137}Cs in the Marshall Islands. Environ Monit Assess 53:363-377.

Sirotkin AN, Shilov VP, Panchenko IYA, et al. 1976. Kinetics of the accumulation excretion of 90Sr and 137Cs in cattle of different ages. S-kh Biol 11(4):538-542.

*Skandalis A, da Cruz AD, Curry J, et al. 1997. Molecular analysis of T-lymphocyte HPRT mutations in individuals exposed to ionizing radiation in Goiania, Brazil. Environ Mol Mutagen 29:107-116.

Skarlou V, Papanicolaou EP, Nobeli C. 1996. Soil to plant transfer of radioactive cesium and its relation to soil and plant properties. Geoderma 72:53-63.

Snipes MB, Boecker BB, McClellan RO. 1983. Retention of monodisperse or polydisperse aluminosilicate particles inhaled by dogs, rats, and mice. Toxicol Appl Pharmacol 69:345-362.

Sora S, Magni GE. 1988. Induction of meiotic chromosomal malsegregation in yeast. Mutat Res 201:375-384.

Spalding BP. 1994. Volatization of cesium-137 from soil with chloride amendments during heating and vitrification. Environ Sci Technol 28:1116-1123.

*Stara JF. 1965. Tissue distribution and excretion of cesium-137 in the guinea pig after administration by three different routes. Health Phys 11:1195-1202.

*Stara JF, Thomas RG. 1963. The tissue distribution and excretion of cesium-137 following inhalation: Preliminary data for rats. AEC Res Dev Rep LF-4.

Stather JW, Phipps AW. 1998. The work of committee 2 of ICRP in developing dose coefficients for the embryo and fetus. Radiat Prot Dosim 79:299-302.

Stather JW, Phipps AW. 1999. The work of committee 2 of ICRP in developing dose coefficients for the embryo and fetus following intakes of radionuclides by the mother. Kerntechnik 64:15-20.

*Steele AK. 1990. Derived concentration factors for caesium-137 in edible species of North Sea fish. Mar Pollut Bull 21(12):591-594.

Steinnes E, Rambaek JP, Hanssen JE. 1992. Large scale multi-element survey of atmospheric deposition using naturally growing moss as biomonitor. Chemosphere 25(5):735-752.

Stetar EA, Boston HL, Larsen IL, et al. 1993. The removal of radioactive cobalt, cesium, and iodine in a conventional municipal wastewater treatment plant. Water Environ Res 65(6):630-639.

Stradling GN, Stather JW, Gray SA, et al. 1989. Assessment of intake of an actinide-bearing dust formed form the pond storage of spent magnox fuel. Radiat Prot Dosim 26(1/4):201-206.

Strebl F, Gerzabek MH, Karg V, et al. 1996. ^{137}Cs-migration in soils and its transfer to roe deer in an Austrian forest stand. Sci Total Environ 181:237-247.

*Strezov A, Stoilova T, Jordanova A, et al. 1999. Determination of caesium and natural radionuclide concentrations in sediments, algae and water in the Bulgarian Black Sea coast. Water Sci Technol 39(8):21-26.

Suchanek TH, Lagunas-Solar MC, Raabe OG, et al. 1996. Radionuclides in fishes and mussels from the Farallon Islands nuclear waste dump site, California. Health Phys 71(2):167-178.

Sugai SF. 1990. Transport and sediment accumulation of ^{210}Pb and ^{137}Cs in two southeast Alaskan Fjords. Estuaries 13(4):380-392.

Sugiura N, Kusama T. 1992. Biokinetics of radionuclides in pregnant mice and compartment models by the system analysis methods. Radiat Prot Dosim 41(2/4):153-156.

9. REFERENCES

*Sun LC, Clinton JH, Kaplan E, et al. 1997. ^{137}Cs exposure in the Marshallese populations: an assessment based on whole-body counting measurements (1989-1994). Health Phys 73:86-99.

Surkova LV, Pogodin RI. 1991. Status forms of occurrence of cesium-137 in soils as a function of distance from the Chernobyl nuclear power plant. Sov Soil Sci 23(10):100-103.

*Szabo B, Patterson E, Scherlag B, et al. 1987. Early after depolarizations induced by Cs^+ are dependent on intra- and extracellular [Ca^{2+}] and [Na^+]. Circulation 76:IV-115.

Szabo B, Sweidan R, Lazzara R. 1985. Cs-induced potentials in purkinje fibers of guinea pig hearts. Fed Proc 44:806.

Szentkuti VL, Breitruck H, Giese W. 1976. [The accumulation of cesium-134 in heart and skeletal muscle of healthy and muscular dystrophic golden hamsters]. Z Versuchstierkd 18(3):121-129. (German).

Takahashi N, Ito M, Ishida S, et al. 1992. Effects of vagal stimulation on cesium-induced early after depolarizations and ventricular arrhythmias in rabbits. Circulation 86:1987-1992.

*Takenaka C, Onda Y, Hamajima Y. 1998. Distribution of cesium-137 in Japanese forest soils: Correlation with the contents of organic carbon. Sci Total Environ 222:193-199.

*Talbot RJ, Newton D, Segal MG. 1993. Gastrointestinal absorption by rats of ^{137}Cs and ^{90}Sr from U_3O_8 fuel particles: Implications for radiation doses to man after a nuclear accident. Radiat Prot Dosim 50(1):39-43.

Tamura T. 1965. Experimental studies on the effect of internal exposure of Cs-137 on the male reproductive organ. The distribution of the Cs-137 in mice body and histological changes of testis by its exposure. II. Jpn J Hyg 20(5):320-328.

Tandon L, Iyengar GV, Parr RM. 1998. A review of radiologically important trace elements in human bones. Appl Radiat Isot 49(8):903-910.

*Tanizaki Y, Shimokawa T, Yamazaki M. 1992. Physico-chemical speciation of trace elements in urban streams by size fractionation. Water Res 26(1):55-63.

*Taskaev E. 1987. Neutron-activation determination of rubidium and caesium in mineral waters. J Radioanal Nucl Chem 118:319-322.

Tempel K. 1997. Influence on species of animal on radionuclide metabolism. Berl Muench Tieraerzl Wochenschr 110:451-455.

Thiry Y, Vanhouche M, Van Der Vaeren P, et al. 1994. Determination of the physico-chemical parameters which influence the Cs availability in forest soils. Sci Total Environ 157:261-265.

*Thomas PA, Gates TE. 1999. Radionuclides in the lichen-caribou-human food chain near uranium mining operations in northern Saskatchewan, Canada. Environ Health Perspect 107(7):527-537.

Thomas RG, Thomas RL. 1968. Long-term retention of ^{137}Cs in the rat. Health Phys 15:83-84.

9. REFERENCES

*Toader M, Vasilache RA, Toader ML, et al. 1996. Cs^{137} transfer from mother to embryos, in the first three years after the Chernobyl accident. In: International Congress, ed. International congress on radiation protection: Ninth international congress of the International Radiation Protection Association, April 14-19, Congress Center Hofburg, Vienna, Austria: proceedings. Seibersdorf, Austria: International Radiation Protection Association. Vol 2, 473-475.

*Tobari I, Matsuda Y, Xiaohung G, et al. 1988. Dose-response relationship for translocation induction in spermatogonia of the crab-eating monkey (Macaca fascicularis) by chronic γ-ray-irradiation. Mutat Res 201:81-87.

*Todorovic D, Popovic D, Djuric G. 1999. Concentration measurements of ^{7}Be and ^{137}Cs in ground level air in the Belgrade city area. Environ Int 25(1):59-66.

Tomioka N, Uchiyama H, Yagi O. 1994. Cesium accumulation and growth characteristics of Rhodococcus erythropolis CS98 and Rhodococcus sp. strain CS402. Appl Environ Microbiol 60(7):2227-2231.

Tondel M, Carlsson G, Hardell L, et al. 1996. Incidence of neoplasms in ages 0-19 Y in parts of Sweden with high ^{137}Cs fallout after the Chernobyl accident. Health Phys 71(6):947-950.

Travnikova IG, Bruk GY, Shutov VN. 1999. Dietary pattern and content of cesium radionuclides in foodstuffs and odies of Braynsk Oblast rural population after the Chernobyl accident. Radiochemistry 41(3):298-301.

TRI97. 2000. Toxics Chemical Release Inventory. National Library of Medicine, National Toxicology Information Program, Bethesda, MD.

*TRI99. 2001. TRI explorer: Providing access to EPA's toxics release inventory data. Washington, DC: Office of Information Analysis and Access. Offices of Environmental Information. U.S. Environmental Protection Agency. Toxic Release Inventory. Http://www.epa.gov/triexplorer/. April 18, 2001.

*Tyler SA, Kretz ND, Norris WP. 1969. Dependence of ^{137}Cs retention on age in the fetal and juvenile beagle dog. AEC Symp Ser 17:191-204.

Uchiyama M. 1977. Estimation of ^{137}Cs body burden in Japanese: I. The ratio of ^{137}Cs in the urine to that in the total excreta following the continuous intake of 137Cs in fall-out. J Radiat Res 18:239-246.

Ugedal O, Jonsson B, Njastad O, et al. 1992. Effects of temperature and body size on radiocaesium retention in brown trout, Salmo trutta. Freshwater Biol 28:165-171.

*Ulsh B, Rademacher S, Whicker FW. 2000. Variations of ^{137}Cs depositions and soil concentrations between alpine and montane soils in northern Colorado. J Environ Radioact 47:57-70.

*UN. 1998. Resolution 1172 (1998). : United Nations. Http://srch0.un.org:80/plweb-cgi/fa...emplateName=predoc.tmpl&setCookie=1. March 23, 2001.

UNSCEAR. 1977. Sources and effects of ionizing radiation: : UNSCEAR 1977 report to the general assembly, with annexes. United Nations Scientific Committee on the Effects of Atomic Radiation. New York: United Nations.

9. REFERENCES

*UNSCEAR. 1996. Sources and effects of ionizing radiation: UNSCEAR 1996 report to the general assembly, with scientific annex. United Nations Scientific Committee on the Effects of Atomic Radiation. New York: United Nations.

*USGS. 1999. Cesium: Mineral commodity summary 1999. U.S. Geological Survey. Http://minerals.usgs.gov/minerals/pubs/commodity/cesium/. August 29, 2000.

*Vandecasteele CM, Van Hees M, Culot JP, et al. 1989. Radiocaesium metabolism in pregnant ewes and their progeny. Sci Total Environ 85:213-223.

Vandelannoote R, Blommaert W, Gijbels R, et al. 1981. Analysis of geothermal waters by spark source mass spectrometry. Fresenius Z Anal Chem 309:291-294.

Vandrot M, Scharbach H, Raylaud G, et al. 1979. Cesium. Encephale 5(4):359-374.

Van Middlesworth L. 1993. ^{129}I and ^{137}Cs fission products in thyroids of animals, 1984-1991. Health Phys 64(1):52-58.

Varskog P, Naeumann R, Steinnes E. 1994. Mobility and plant availability of radioactive Cs in natural soil in relation to stable Cs, other alkali elements and soil fertility. J Environ Radioact 22:43-53.

Vasilenko II. 1991. Carcinogenic hazards of radioactive cesium. Vopr Onkol 37(4):394-400.

*Vertacnik A, Prohic E, Juracic M, et al. 1997. Selected element concentrations in alluvial sediments under garbage disposal site (Zagreb, Croatia). Water Res 31(6):1421-1429.

*Vieira I, Sonnier M, Cresteil T. 1996. Developmental expression of *CYP2E1* in the human liver: Hypermethylation control of gene expression during the neonatal period. Eur J Biochem 238:476-483.

Voigt G, Muller H, Paretzke HG, et al. 1993. ^{137}Cs transfer after Chernobyl from fodder into chicken meat and eggs. Health Phys 65(2):141-146.

Wang CC. 1980. Treatment of malignant tumors of the nasopharynx. Otolaryngol Clin North Am 13(3):477-481.

Wang S. 1988. The dependence of thermoluminescence response and glow curve structure of LiF (Mg, Cu, P) TL materials on Mg, Cu, P dopants concentration. Radiat Prot Dosim 25:133-136.

Ward NI, Savage JM. 1994. Metal dispersion and transportational activities using food crops as biomonitors. Sci Total Environ 146/147:309-319.

Warren CJ, Dudas MJ. 1988. Leaching behaviour of selected trace elements in chemically weathered alkaline fly ash. Sci Total Environ 76:229-246.

*Watson WS. 1987. Human radiocesium levels in Scotland. In: Hamphill DD, ed. Trace substances in environmental health-XXI: Proceedings of the University of Missouri's 21st annual conference on trace substances in environmental health. Columbia, Missouri: University of Missouri, 232-236.

Watson WS, Sumner DJ, Baker JR, et al. 1999. Radionuclides in seals and porpoises in the coastal waters around the UK. Sci Total Environ 234:1-13.

9. REFERENCES

Wellner U. 1989. A pharmacokinetic approach to investigate the uptake of ^{137}Cs by children after the reactor accident in Chernobyl. Nucl Med Commun 28:243-246.

*West JR, Smith HW, Chasis H. 1948. Glomerular filtration rate, effective renal blood flow, and maximal tubular excretory capacity in infancy. J Pediatr 32:10-18.

*WHO. 1983. Selected radionuclides: Tritium, carbon-14, krypton-85, strontium-90, iodine, caesium-137, radon, plutonium. Environmental Health Criteria 25. Geneva: World Health Organization.

*Widdowson EM, Dickerson JWT. 1964. Chemical composition of the body. In: Comar CL, Bronner F, eds. Mineral metabolism: An advanced treatise. Vol II: The elements Part A. New York: Academic Press.

Wiley LM, Baulch JE, Raabe OG, et al. 1997. Impaired cell proliferation in mice that persists across at least two generations after paternal irradiation. Radiat Res 148:145-151.

*Willey NJ, Martin MH. 1995. Annual patterns of Cs-133 concentrations in British upland vegetation. Chemosphere 30(4):717-724.

Willey NJ, Martin MH. 1997. A comparison of stable caesium uptake by six grass species of contrasting growth strategy. Environ Pollut 95(3):311-317.

Wyoff MH. 1971. Distribution of ^{134}Cs in the conceptus of the pregnant rat. Radiat Res 47:628-634.

Yamagata N, Iwashima K. 1966. Distribution of cesium and rubidium in human blood. Nature 211:528-529.

Yamamoto N, Kasai M. 1982. Inhibition of a voltage-dependent cation channel in sarcoplasmic reticulum vesicles by cesium studied by using a potential-sensitive cyanine dye. Biochim Biophys Acta 692:89-96.

Yanagi T, Tatanabe M, Yamamoto K. 1989. Sorption behavior of cesium and strontium ions on mixtures of clay sorbents. 26(9):861-864.

*Yasuda H, Uchida S, Muramatsu Y, et al. 1995. Sorption of manganese, cobalt, zinc, strontium, and cesium onto agricultural soils: Statistical analysis on effects of soil properties. Water Air Soil Pollut 83:85-96.

Yoshida S, Muramatsu Y. 1994. Accumulation of radiocesium in basidiomycetes collected from Japanese forests. Sci Total Environ 157:197-205.

Yoshimoto Y, Kato H, Schull WJ. 1988. Risk of cancer among children exposed in utero to A-bomb radiations, 1950-1984. Lancet 2(8612):665-669.

*Yoshioka M, Kitahar K, Keida Y, et al. 1976. Strontium-90 and cesium-137 in human placenta. Acta Med Univ Kagoshima 18:113-117.

Zapoliskaya NA, Lavrentlev LF, Zhorno LYA. 1973. Carcinogenic effect in continuous uptake of some radionuclides and chronic external irradiation. Vopr Onkol 19(9):80-85.

Zhorno L, Iakovleva NG. 1974. Radiation injury to the sexual and somatic cells of rats with a chronic intake of radionuclides. Radiobiologiia 14(1):123-125.

Zhu S, Xia F, Yang W. 1997a. [Comparison of chromosomal aberration induced by signal radionuclide ^{134}Cs in somatic and germ cells.] Zhong Fan Yixue Yu Fanghu Zazhi 17(4):239-242. (Chinese).

Zhu S, Xia F, Yang W. 1997b. [Effect of chromosomal aberrations in spermatogonia induced by ^{134}Cs retention in germ cells.] Fushe Fanghu 17(3):208-211. (Chinese).

*Ziegler EE, Edwards BB, Jensen RL, et al. 1978. Absorption and retention of lead by infants. Pediatr Res 12:29-34.

*Zundel WS, Tyler FH, Mays CW, et al. 1969. Short half-times of caesium-137 in pregnant women. Nature 221:89-90.

Zvonova IA, Balonov MI. 1993. Radioiodine dosimetry and prediction of consequences of thyroid exposure of the Russian population following the Chernobyl accident. In: Merwin SE, Balonov MI, eds. The Chernobyl papers: Doses to the Soviet population and early health effects studies. Richland, WA: Research Enterprises, Inc. Vol I. 71-125.

10. GLOSSARY

Absorbed Dose, Chemical—The amount of a substance that is either absorbed into the body or placed in contact with the skin. For oral or inhalation routes, this is normally the product of the intake quantity and the uptake fraction divided by the body weight and, if appropriate, the time, expressed as mg/kg for a single intake or mg/kg/day for multiple intakes. For dermal exposure, this is the amount of material applied to the skin, and is normally divided by the body mass and expressed as mg/kg.

Absorbed Dose, Radiation—The mean energy imparted to the irradiated medium, per unit mass, by ionizing radiation. Units: rad (rad), gray (Gy).

Absorbed Fraction—A term used in internal dosimetry. It is that fraction of the photon energy (emitted within a specified volume of material) which is absorbed by the volume. The absorbed fraction depends on the source distribution, the photon energy, and the size, shape and composition of the volume.

Absorption—The process by which a chemical penetrates the exchange boundaries of an organism after contact, or the process by which radiation imparts some or all of its energy to any material through which it passes.

Absorption Coefficient—Fractional absorption of the energy of an unscattered beam of x- or gamma-radiation per unit thickness (linear absorption coefficient), per unit mass (mass absorption coefficient), or per atom (atomic absorption coefficient) of absorber, due to transfer of energy to the absorber. The total absorption coefficient is the sum of individual energy absorption processes (see Compton Effect, Photoelectric Effect, and Pair Production).

Absorption Coefficient, Linear—A factor expressing the fraction of a beam of x- or gamma radiation absorbed in a unit thickness of material. In the expression $I=I_o e^{-\mu x}$, I_o is the initial intensity, I the intensity of the beam after passage through a thickness of the material x, and μ is the linear absorption coefficient.

Absorption Coefficient, Mass—The linear absorption coefficient per cm divided by the density of the absorber in grams per cubic centimeter. It is frequently expressed as μ/ρ, where μ is the linear absorption coefficient and ρ the absorber density.

Absorption Ratio, Differential—Ratio of concentration of a nuclide in a given organ or tissue to the concentration that would be obtained if the same administered quantity of this nuclide were uniformly distributed throughout the body.

Activation—The process of making a material radioactive by bombardment with neutrons or protons.

Activity—The number of radioactive nuclear transformations occurring in a material per unit time (see Curie, Becquerel). The term for activity per unit mass is specific activity.

Activity Median Aerodynamic Diameter (AMAD)—The diameter of a unit-density sphere with the same terminal settling velocity in air as that of the aerosol particle whose activity is the median for the entire size distribution of the aerosol.

Acute Exposure, Chemical—Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Acute Exposure, Radiation—The absorption of a relatively large amount of radiation (or intake of a radioactive material) over a short period of time.

Acute Radiation Syndrome—The symptoms which taken together characterize a person suffering from the effects of intense radiation. The effects occur within hours or days.

Ad libitum—Available in excess and freely accessible.

Adsorption Coefficient (K_{oc})—The ratio of the amount of a chemical adsorbed per unit surface area or per unit weight of organic carbon of a specific particle size in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (K_d)—See Distribution Coefficient

Alpha Particle—A positively charged particle ejected spontaneously from the nuclei of some radioactive elements. It is identical to a helium nucleus, i.e., 2 neutrons and two protons, with a mass number of 4 and an electrostatic charge of +2.

Alpha Track—The track of ionized atoms (pattern of ionization) left in a medium by an alpha particle that has traveled through the medium.

Annihilation (Positron-Electron)—An interaction between a positive and a negative electron in which they both disappear; their rest mass, being converted into electromagnetic radiation (called annihilation radiation) with two 0.51 MeV gamma photons emitted at an angle of 180E to each other.

Atom—The smallest particle of an element that cannot be divided or broken up by chemical means. It consists of a central core called the *nucleus*, which contains *protons* and *neutrons* and an outer shell of *electrons*.

Atomic Mass (u)—The mass of a neutral atom of a nuclide, usually expressed in terms of "atomic mass units." The "atomic mass unit" is one-twelfth the mass of one neutral atom of carbon-12; equivalent to 1.6604×10^{-24} g.

Atomic Number—The number of protons in the nucleus of an atom. The "effective atomic number" is calculated from the composition and atomic numbers of a compound or mixture. An element of this atomic number would interact with photons in the same way as the compound or mixture. (Symbol: Z).

Atomic Mass Number—See Mass Number.

Atomic Weight—The weighted mean of the masses of the neutral isotopes of an element expressed in atomic mass units.

Attenuation—A process by which a beam from a source of radiation is reduced in intensity by absorption and scattering when passing through some material.

Attenuation Coefficient—The fractional reduction in the intensity of a beam of radiation as it passes through an absorbing medium. It may be expressed as reduction per unit distance, per unit mass thickness, or per atom, and is called the linear, mass, or atomic attenuation coefficient, respectively.

Auger Effect—The emission of an electron from the extranuclear portion of an excited atom when the atom undergoes a transition to a less excited state.

Background Radiation—The amount of radiation to which a member of the general population is exposed from natural sources, such as terrestrial radiation from naturally occurring radionuclides in the soil, cosmic radiation originating from outer space, and naturally occurring radionuclides deposited in the human body.

Becquerel (Bq)—International System of Units unit of activity and equals that quantity of radioactive material in which one transformation (disintegration) occurs per second (see Units).

Beta Particle—An electron that is emitted from the nucleus of an atom during one type of radioactive transformation. A beta particle has a mass and charge equal in magnitude to that of the electron. The charge may be either +1 or -1. Beta particles with +1 charges are called positrons (symbolized β^+), and beta particles with -1 charges are called negatrons (symbolized β^-).

Biological Half-time—The time required for a biological system, such as that of a human, to eliminate by natural process half of the amount of a substance (such as a chemical substance, either stable or radioactive) that has entered it.

Bioconcentration Factor (BCF)—The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Biologic Effectiveness of Radiation—See Relative Biological Effectiveness.

Biomarkers—Broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility.

Body Burden, Chemical—The total amount of a chemical found in an animal or human body.

Body Burden, Radioactivity—The amount of radioactive material found in an animal or human body.

Bone Seeker—Any compound or ion which migrates in the body and preferentially deposits into bone.

Branching—The occurrence of two or more modes by which a radionuclide can undergo radioactive decay. For example, ^{214}Bi can undergo alpha or beta minus decay, ^{64}Cu can undergo beta minus, beta plus, or electron capture decay. An individual atom of a nuclide exhibiting branching disintegrates by one mode only. The fraction disintegrating by a particular mode is the "branching fraction" for that mode. The "branching ratio" is the ratio of two specified branching fractions (also called multiple disintegration).

Bremsstrahlung—X rays that are produced when a charged particle accelerates (speeds up, slows down, or changes direction) in the strong field of a nucleus.

Buildup Factor—The ratio of the radiation intensity, including both primary and scattered radiation, to the intensity of the primary (unscattered) radiation.

Cancer Effect Level (CEL)—The lowest dose of chemical or radiation in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Capture, Electron—A mode of radioactive decay involving the capture of an orbital electron by its nucleus. Capture from a particular electron shell, e.g., K or L shells, is designated as "K-electron capture" or "L-electron capture."

Capture, K-Electron—Electron capture from the K shell by the nucleus of the atom. Also loosely used to designate any orbital electron capture process.

Carcinogen—A chemical or radiation that is capable of inducing cancer.

Carcinoma—Malignant neoplasm composed of epithelial cells, regardless of their derivation.

Case-Control Study—A type of epidemiological study which examines the relationship between a particular outcome (disease or condition) and a variety of potential causative agents (such as toxic chemicals). In a case-controlled study, a group of people with a specified and well-defined outcome is identified and compared to a similar group of people without outcome.

Case Report—Describes a single individual with a particular disease or exposure. These may suggest some potential topics for scientific research but are not actual research studies.

Cataract—A clouding of the crystalline lens of the eye which obstructs the passage of light.

Ceiling Value—A concentration of a substance that should not be exceeded, even temporarily.

Charged Particle—A nuclear particle, atom, or molecule carrying a positive or negative charge.

Chronic Exposure—Exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Cohort Study—A type of epidemiological study of a specific group or groups of people who have had a common insult (e.g., exposure to an agent suspected of causing disease or a common disease) and are followed forward from exposure to outcome. At least one exposed group is compared to one unexposed group.

Collective dose—The sum of the individual doses received in a given period of time by a specified population from exposure to a specified source of radiation. Collective dose is expressed in units such as man-rem and person-sievert.

Compton Effect—An attenuation process observed for x- or gamma radiation in which an incident photon interacts with an orbital electron of an atom to produce a recoil electron and a scattered photon whose energy is less than the incident photon.

Containment—The confinement of a chemical or radioactive substance in such a way that it is prevented from being dispersed from its container or into the environment, or is released only at a specified rate.

Contamination—Deposition of a stable or radioactive substance in any place where it is not desired.

Cosmic Rays—High-energy particulate and electromagnetic radiations which originate outside the earth's atmosphere.

Count (Radiation Measurements)—The external indication of a radiation-measuring device designed to enumerate ionizing events. It refers to a single detected event. The term "count rate" refers to the total number registered in a given period of time. The term is sometimes erroneously used to designate a disintegration, ionizing event, or voltage pulse.

Counter, Gas-flow Proportional (GPC)—β-particles are detected by ionization of the counter gas which results in an electrical impulse at an anode wire. If a sufficient amount of radiostrontium is present and the ionization efficiency is calibrated, the quantity of radiostrontium can be determined.

Counter, Geiger-Mueller (GM counter)—Highly sensitive, gas-filled radiation-measuring device to detect (count) individual photons or particulate radiation.

Counter, Scintillation—The combination of phosphor, photomultiplier tube, and associated circuits for counting light emissions produced in the phosphors by ionizing radiation. Scintillation counters generally are more sensitive than GM counters for gamma radiation.

Counting, Cerenkov — Relatively energetic β-particles pass through a transparent medium of high refractive index and a highly-directional, bluish-white light ("Cerenkov" light) is emitted. This light is detected using liquid scintillation counting equipment.

Cross-sectional Study—A type of epidemiological study of a group or groups which examines the relationship between exposure and outcome to a chemical or to chemicals at one point in time.

Curie (Ci)—A unit of radioactivity. One curie equals that quantity of radioactive material in which there are 3.7×10^{10} nuclear transformations per second. The activity of 1 gram of radium is approximately 1 Ci.

Attocurie (aCi)—One-thousandth of a femtocurie (3.7×10^{-8} disintegrations per second).
Femtocurie (fCi)—One-billionth of a microcurie (3.7×10^{-5} disintegrations per second).
Megacurie (MCi)—One million curies (3.7×10^{16} disintegrations per sec).
Microcurie (μCi)—One-millionth of a curie (3.7×10^{4} disintegrations per sec).
Millicurie (mCi)—One-thousandth of a curie (3.7×10^{7} disintegrations per sec).
Nanocurie (nCi)—One-billionth of a curie (3.7×10^{1} disintegrations per sec).
Picocurie (pCi)—One-millionth of a microcurie (3.7×10^{-2} disintegrations per second.

Daughter Products—See Progeny and Decay Product

Decay, Radioactive—Transformation of the nucleus of an unstable nuclide by spontaneous emission of charged particles and/or photons (see Disintegration).

Decay Chain or Decay Series—A sequence of radioactive decays (transformations) beginning with one nucleus. The initial nucleus, the parent, decays into a daughter or progeny nucleus that differs from the first by whatever particles were emitted during the decay. If further decays take place, the subsequent nuclei are also usually called daughters or progeny. Sometimes, to distinguish the sequence, the daughter of the first daughter is called the granddaughter, etc.

Decay Constant (λ)—The fraction of the number of atoms of a radioactive nuclide which decay in unit time (see Disintegration Constant).

Decay Product, Daughter Product, Progeny—A new nuclide formed as a result of radioactive decay. A nuclide resulting from the radioactive transformation of a radionuclide, formed either directly or as the result of successive transformations in a radioactive series. A decay product (daughter product or progeny) may be either radioactive or stable.

Delta Ray—Electron removed from an atom during the process of ionization (also called secondary electron). Delta rays cause a track of ionizations along their path.

Depleted uranium (DU)—Uranium having a percentage of ^{235}U smaller than the 0.7% found in natural uranium. It is obtained as a by-product from ^{235}U enrichment.

Developmental Toxicity—The occurrence of adverse effects on the developing organism that may result from exposure to a chemical or radiation prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Disintegration Constant—Synonymous with decay constant. The fraction of the number of atoms of a radioactive material that decays per unit time (see Decay Constant.)

Disintegration, Nuclear—A spontaneous nuclear transformation (radioactivity) characterized by the emission of energy and/or mass from the nucleus. When large numbers of nuclei are involved, the process is characterized by a definite half-life (see Transformation, Nuclear).

Distribution coefficient (K_d)—Describes the distribution of a chemical between the solid and aqueous phase at thermodynamic equilibrium, is given as follows:

$$K_d = \frac{[C]_s}{[C]_w}\ ,\ \text{Units} = (\text{L solution})/(\text{kg solid}),$$

where $[C]_s$ is the concentration of the chemical associated with the solid phase in units of (mg)/(kg solid), and $[C]_w$ is the concentration of the chemical in the aqueous phase in units of (mg)/(L solution). As the magnitude of K_d decreases, the potential mobility of the chemical to groundwater systems increases and vice versa .

Dose—A general term denoting the quantity of a substance, radiation, or energy absorbed. For special purposes it must be appropriately qualified. If unqualified, it refers to radiation absorbed dose.

> **Absorbed Dose**—The energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the rad. One rad equals 100 ergs per gram. In SI units, the absorbed dose is the gray which is 1 J/kg (see Rad).
>
> **Cumulative Dose (Radiation)**—The total dose resulting from repeated or continuous exposures to radiation.
>
> **Dose Assessment**—An estimate of the radiation dose to an individual or a population group usually by means of predictive modeling techniques, sometimes supplemented by the results of measurement.

Dose Equivalent (DE)—A quantity used in radiation safety practice to account for the relative biological effectiveness of the several types of radiation. It expresses all radiations on a common scale for calculating the effective absorbed dose. It is defined as the product of the absorbed dose in rad and certain modifying factors. (The unit of dose equivalent is the rem. In SI units, the dose equivalent is the sievert, which equals 100 rem.)

Dose, Radiation—The amount of energy imparted to matter by ionizing radiation per unit mass of the matter, usually expressed as the unit rad, or in SI units, the gray. 100 rad ' 1 gray (Gy) (see Absorbed Dose).

Maximum Permissible Dose Equivalent (MPD)—The greatest dose equivalent that a person or specified part thereof shall be allowed to receive in a given period of time.

Median Lethal Dose (MLD)—Dose of radiation required to kill, within a specified period (usually 30 days), 50% of the individuals in a large group of animals or organisms. Also called the LD_{50}, or $LD_{50/30}$ if for 30 days..

Threshold Dose—The minimum absorbed dose that will produce a detectable degree of any given effect.

Tissue Dose—Absorbed dose received by tissue in the region of interest, expressed in rad (see Dose, Gray, and Rad).

Dose, Fractionation—A method of administering therapeutic radiation in which relatively small doses are given daily or at longer intervals.

Dose, Protraction—A method of administering therapeutic radiation by delivering it continuously over a relatively long period at a low dose rate.

Dose Rate—Absorbed dose delivered per unit time.

Dose-Response Relationship—The quantitative relationship between the amount of exposure to a toxicant and the incidence of the adverse effects.

Dosimetry—Quantification of radiation doses to cells, tissues, organs, individuals or populations resulting from specified exposures.

Early Effects (of radiation exposure)—Effects that appear within 60 days of an acute exposure.

Electron—A stable elementary particle having an electric charge equal to $\pm 1.60210 \times 10^{-19}$ C (Coulombs) and a rest mass equal to 9.1091×10^{-31} kg. A positron is a positively charged "electron" (see Positron).

Electron Volt—A unit of energy equivalent to the energy gained by an electron in passing through a potential difference of one volt. Larger multiple units of the electron volt are frequently used: keV for thousand or kilo electron volts; MeV for million or mega electron volts (eV). 1 eV=1.6×10^{-12} erg.

Embryotoxicity and Fetotoxicity—Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and *in utero* death.

Energy—Capacity for doing work. "Potential energy" is the energy inherent in a mass because of its spatial relation to other masses. "Kinetic energy" is the energy possessed by a mass because of its motion (SI unit: joules):

Binding Energy (Electron)—The amount of energy that must be expended to remove an electron from an atom.

Binding Energy (Nuclear)—The energy represented by the difference in mass between the sum of the component parts and the actual mass of the nucleus. It represents the amount of energy that must be expended to break a nucleus into its component neutrons and protons.

Excitation Energy—The energy required to change a system from its ground state to an excited state. Each different excited state has a different excitation energy.

Ionizing Energy—The energy required to knock an electron out of an atom. The average energy lost by electrons or beta particles in producing an ion pair in air or in soft tissue is about 34 eV.

Radiant Energy—The energy of electromagnetic radiation, such as radio waves, visible light, x and gamma rays.

Enriched Material—Material in which the relative amount of one or more isotopes of a constituent has been increased.

Enriched Uranium—Uranium in which the abundance of the ^{235}U isotope is increased above normal.

Enrichment, Isotopic—An isotopic separation process by which the relative abundances of the isotopes of a given element are altered, thus producing a form of the element that has been enriched in one or more isotopes and depleted in others. In uranium enrichment, the percentage of uranium-235 in natural uranium can be increased from 0.7% to >90% in a gaseous diffusion process based on the different thermal velocities of the constituents of natural uranium (^{234}U, ^{235}U, ^{238}U) in the molecular form UF_6.

EPA Health Advisory—An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Epidemiology—Refers to the investigation of factors that determine the frequency and distribution of disease or other health-related conditions within a defined human population during a specified period.

Equilibrium, Radioactive—In a radioactive series, the state which prevails when the ratios between the activities of two or more successive members of the series remains constant.

Secular Equilibrium—If a parent element has a very much longer half-life than the daughters (so there is not appreciable change in its amount in the time interval required for later products to attain equilibrium) then, after equilibrium is reached, equal numbers of atoms of all members of the series disintegrate in unit time. This condition is never exactly attained, but is essentially established in such a case as ^{226}Ra and its transformation series to stable ^{206}Pb. The half-life of ^{226}Ra is about 1,600 years; of ^{222}Rn, approximately 3.82 days, and of each of the subsequent members, a few minutes. After about a month, essentially the equilibrium amount of radon is present; then (and for a long time) all members of the series disintegrate the same number of atoms per unit time. At this time, the activity of the daughter is equal to the activity of the parent.

Transient Equilibrium—If the half-life of the parent is short enough so the quantity present decreases appreciably during the period under consideration, but is still longer than that of successive members of the series, a stage of equilibrium will be reached after which all members of the series decrease in activity exponentially with the period of the parent. At this time, the ratio of the parent activity to the daughter activity is constant.

Equilibrium, Electron—The condition in a radiation field where the energy of the electrons entering a volume equals the energy of the electrons leaving that volume.

Equilibrium Fraction (F)—In radon-radon daughter equilibrium, the parents and daughters have equal radioactivity, that is, as many decay into a specific nuclide as decay out. However, if fresh radon is continually entering a volume of air or if daughters are lost by processes other than radioactive decay, e.g., plate out or migration out of the volume, a disequilibrium develops. The equilibrium fraction is a measure of the degree of equilibrium/disequilibrium. The equilibrium fraction is used to estimate working levels based on measurement of radon only. For radon, 1 working-level concentration is defined at 100 pCi of radon in equilibrium with its 4 successive progeny in 1 liter of air. Thus, 100 pCi/L radon at 50% equilibrium is 0.5 WL.

Excitation—The addition of energy to a system, thereby transferring it from its ground state to an excited state. Excitation of a nucleus, an atom, or a molecule can result from absorption of photons or from inelastic collisions with other particles. The excited state of an atom is an unstable or metastable state and will return to ground state by radiation of the excess energy.

Exposure (Chemical)—Contact of an organism with a chemical or physical agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, gut) and available for absorption.

Exposure (Radiation)—Being exposed to ionizing radiation or to a radioactive material. A measure of the ionization produced in air by x or gamma radiation; the sum of the electric charges on all ions of one sign produced in air when all electrons liberated by photons in a volume of air are completely stopped in air (dQ), divided by the mass of the air in the volume (dm). The unit of exposure in air is the roentgen, or coulomb per kilogram (SI units). One roentgen is equal to 2.58×10^{-4} coulomb per kilogram (C/kg).

Fission, Nuclear—A nuclear transformation characterized by the splitting of a nucleus into at least two other nuclei and several neutrons, and is accompanied by the release of a relatively large amount of energy.

Gamma Ray, Penetrating—Short wavelength electromagnetic radiation of nuclear origin.

Genetic Effect of Radiation—Inheritable change, chiefly mutations, produced by the absorption of ionizing radiation by germ cells. Genetic effects have not been observed in any human population exposed at any dose level.

Genotoxicity—A specific adverse effect on the genome of living cells that, upon the duplication of affected cells, can be expressed as a mutagenic, clastogenic or carcinogenic event because of specific alteration of the molecular structure of the genome.

Gray (Gy)—SI unit of absorbed dose, 1 J/kg. One gray equals 100 rad (see Units).

Half-life, Radioactive—Time required for a radioactive substance to lose 50% of its activity by decay. Each radio-nuclide has a unique physical half-life. Known also as physical half-time and symbolized as T_r or T_{rad}.

Half-life, Effective—See Half-Time, Effective.

Half-time, Biological—Time required for an organ, tissue, or the whole body to eliminate one-half of any absorbed substance by regular processes of elimination. This is the same for both stable and radioactive isotopes of a particular element, and is sometimes referred to as half-time, symbolized as t_{biol} or T_b.

Half-time, Effective—Time required for a radioactive element in an organ, tissue, or the whole body to be diminished 50% as a result of the combined action of radioactive decay and biological elimination, symbolized as T_e or T_{eff}.

$$\textit{Effective Half\&time} = \frac{\textit{Biological half\&time x Radioactive half\&life}}{\textit{Biological half\&time \% Radioactive half\&life}}$$

Immediately Dangerous to Life or Health (IDLH)—The maximum environmental concentration of a contaminant from which one could escape within 30 minutes without any escape-impairing symptoms or irreversible health effects.

Immunologic Toxicity—The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

Immunological Effects—Functional changes in the immune response.

In Vitro—Isolated from the living organism and artificially maintained, as in a test tube. Literally, “in glass.”

In Vivo—Occurring within the living organism. Literally, “in life.”

Intensity—Amount of energy per unit time passing through a unit area perpendicular to the line of propagation at the point in question.

Intermediate Exposure—Exposure to a chemical for a duration of 15–364 days, as specified in the Toxicological Profiles.

Internal Conversion—Process in which a gamma ray knocks an electron out of the same atom from which the gamma ray was emitted. The ratio of the number of internal conversion electrons to the number of gamma quanta emitted in the de-excitation of the nucleus is called the "conversion ratio."

Ion—Atomic particle, atom or chemical radical bearing a net electrical charge, either negative or positive.

Ion Pair—Two particles of opposite charge, usually referring to the electron and positive atomic or molecular residue resulting after the interaction of ionizing radiation with the orbital electrons of atoms.

Ionization—The process by which a neutral atom or molecule acquires a positive or negative charge.

> **Primary Ionization**—(1) In collision theory: the ionization produced by the primary particles as contrasted to the "total ionization" which includes the "secondary ionization" produced by delta

rays. (2) In counter tubes: the total ionization produced by incident radiation without gas amplification.

Specific Ionization—Number of ion pairs per unit length of path of ionizing radiation in a medium; e.g., per centimeter of air or per micrometer of tissue.

Total Ionization—The total electric charge of one sign on the ions produced by radiation in the process of losing its kinetic energy. For a given gas, the total ionization is closely proportional to the initial ionization and is nearly independent of the nature of the ionizing radiation. It is frequently used as a measure of absorption of radiation energy.

Ionization Density—Number of ion pairs per unit volume.

Ionization Path (Track)—The trail of ion pairs produced by an ionizing particle in its passage through matter.

Ionizing Radiation—Any radiation capable of knocking electrons out of atoms and producing ions. Examples: alpha, beta, gamma and x rays, and neutrons.

Isobars—Nuclides having the same mass number but different atomic numbers.

Isomers—Nuclides having the same number of neutrons and protons but capable of existing, for a measurable time, in different quantum states with different energies and radioactive properties. Commonly the isomer of higher energy decays to one with lower energy by the process of isomeric transition.

Isotopes—Nuclides having the same number of protons in their nuclei, and hence the same atomic number, but differing in the number of neutrons, and therefore in the mass number. Identical chemical properties exist in isotopes of a particular element. The term should not be used as a synonym for nuclide because isotopes refer specifically to different nuclei of the same element.

Stable Isotope—A nonradioactive isotope of an element.

Kerma (k)—A measure of the kinetic energy transferred from gamma rays or neutrons to a unit mass of absorbing medium in the initial collision between the radiation and the absorber atoms. The SI unit is J/kg. The special name of this unit is the rad (traditional system of units) or Gray (SI).

Joule—The S.I. unit for work and energy. It is equal to the work done by raising a mass of one newton through a distance of one meter (J = Nm), which corresponds to about 0.7 ft-pound.

Labeled Compound—A compound containing one or more radioactive atoms intentionally added to its structure. By observations of radioactivity or isotopic composition, this compound or its fragments may be followed through physical, chemical, or biological processes.

Late Effects (of radiation exposure)—Effects which appear 60 days or more following an acute exposure.

$LD_{50/30}$—The dose of a chemical or radiation expected to cause 50% mortality in those exposed within 30 days. For radiation, this is about 350 rad (3.5 gray) received by humans over a short period of time.

Lethal Concentration$_{(LO)}$ (LC_{LO})—The lowest concentration of a chemical in air that has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC_{50})—A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population within a specified time, usually 30 days.

Lethal Dose$_{(L0)}$ (LD_{L0})—The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals within a specified time, usually 30 days.

Lethal Dose$_{(50)}$ (LD_{50})—The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$ (LT_{50})—A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Linear Energy Transfer (LET)—A measure of the energy that a charged particle transfers to a material per unit path length.

> **Low-LET**—Energy transfer characteristic of light charged particles such as electrons produced by x and gamma rays where the distance between ionizing events is large on the scale of a cellular nucleus.
>
> **High-LET**—Energy transfer characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus.
>
> **Average LET**—The energy of a charged particle divided by the length of the path over which it deposits all its energy in a material.

Lowest-Observed-Adverse-Effect Level (LOAEL)—The lowest dose of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Lung Clearance Class (fast, F; medium, M; slow, S)—A classification scheme for inhaled material according to its rate of clearance from the pulmonary region of the lungs to the blood and the gastrointestinal tract.

Lymphoreticular Effects—Represent morphological effects involving lymphatic tissues such as the lymph nodes, spleen, and thymus.

Malformations—Permanent structural changes that may adversely affect survival, development, or function.

Mass Numbers (A)—The number of nucleons (protons and neutrons) in the nucleus of an atom.

Minimal Risk Level—An estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.

Morbidity—State of being diseased; morbidity rate is the incidence or prevalence of disease in a specific population.

Mutagen—A substance that causes changes (mutations) in the genetic material in a cell. Mutations can lead to birth defects, miscarriages, or cancer.

Necropsy—The gross examination of the organs and tissues of a dead body to determine the cause of death or pathological conditions.

Neurotoxicity—The occurrence of adverse effects on the nervous system following exposure to a substance.

Neutrino (ν)—A neutral particle of infinitesimally small rest mass emitted during beta plus or beta minus decay. This particle accounts for conservation of energy in beta plus and beta minus decays. It plays no role in damage from radiation.

No-Observed-Adverse-Effect Level (NOAEL)—The dose of a substance at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Nuclear reactor—A power plant that heats water by using nuclear reactions instead of burning coal, oil, or natural gas. All of these sources of energy simply heat water and use the steam which is produced to turn turbines that make electricity or propel a ship.

Nucleon—Common name for a constituent particle of the nucleus. Applied to a proton or neutron.

Nuclide—A species of atom characterized by the constitution of its nucleus. The nuclear constitution is specified by the number of protons (Z), number of neutrons (N), and energy content; or, alternatively, by the atomic number (Z), mass number A ' (N+Z), and atomic mass. To be regarded as a distinct nuclide, the atom must be capable of existing for a measurable time. Thus, nuclear isomers are separate nuclides, whereas promptly decaying excited nuclear states and unstable intermediates in nuclear reactions are not so considered.

Octanol-Water Partition Coefficient (K_{ow})—The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Odds Ratio (OR)—A means of measuring the association between an exposure (such as toxic substances and a disease or condition) which represents the best estimate of relative risk (risk as a ratio of the incidence among subjects exposed to a particular risk factor divided by the incidence among subjects who were not exposed to the risk factor). An odds ratio of greater than 1 is considered to indicate greater risk of disease in the exposed group compared to the unexposed.

Pair Production—An absorption process for x- and gamma radiation in which the incident photon is absorbed in the vicinity of the nucleus of the absorbing atom, with subsequent production of an electron and positron pair (see annihilation). This reaction can only occur for incident photon energies exceeding 1.02 MeV.

Parent—A radionuclide which, upon disintegration, yields a new nuclide, either directly or as a later member of a radioactive series.

Permissible Exposure Limit (PEL)—A maximum allowable atmospheric level of a substance in workplace air averaged over an 8-hour shift.

Pharmacokinetics—The science of quantitatively predicting the fate (disposition) of an exogenous substance in an organism. Utilizing computational techniques, it provides the means of studying the absorption, distribution, metabolism and excretion of chemicals by the body.

Pharmacokinetic Model—A set of equations that can be used to describe the time course of a parent chemical or metabolite in an animal system. There are two types of pharmacokinetic models: data-based and physiologically-based. A data-based model divides the animal system into a series of compartments which, in general, do not represent real, identifiable anatomic regions of the body whereas the physiologically-based model compartments represent real anatomic regions of the body.

Physiologically Based Pharmacodynamic (PBPD) Model—A type of physiologically-based dose-response model which quantitatively describes the relationship between target tissue dose and toxic end points. These models advance the importance of physiologically based models in that they clearly describe the biological effect (response) produced by the system following exposure to an exogenous substance.

Physiologically Based Pharmacokinetic (PBPK) Model—A model comprising a series of compartments representing organs or tissue groups with realistic weights and blood flows. These models require a variety of physiological information: tissue volumes, blood flow rates to tissues, cardiac output, alveolar ventilation rates and, possibly membrane permeabilities. The models also utilize biochemical information such as air/blood partition coefficients, and metabolic parameters. PBPK models are also called biologically based tissue dosimetry models.

Photon—A quantum of electromagnetic energy (E) whose value is the product of its frequency (ν) in hertz and Planck's constant (h). The equation is: $E = h\nu$.

Photoelectric Effect—An attenuation process observed for x and gamma radiation in which an incident photon interacts with a tightly bound inner orbital electron of an atom delivering all of its energy to knock the electron out of the atom. The incident photon disappears in the process.

Positron—A positively charged electron.

Potential, Ionization—The energy expressed as electron volts (eV) necessary to separate one electron from an atom, resulting in the formation of an ion pair.

Power, Stopping—A measure of the ability of a material to absorb energy from an ionizing particle passing through it; the greater the stopping power, the greater the energy absorbing ability (see Linear Energy Transfer).

Progeny—The decay product or products resulting after a radioactive decay or a series of radioactive decays. The progeny can also be radioactive, and the chain continues until a stable nuclide is formed.

Proton—Elementary nuclear particle with a positive electric charge equal numerically to the charge of the electron and a rest mass of 1.007 mass units.

Quality—A term describing the distribution of the energy deposited by a particle along its track; radiations that produce different densities of ionization per unit intensity are said to have different "qualities."

Quality Factor (Q)—The linear-energy-transfer-dependent factor by which absorbed doses are multiplied to obtain (for radiation protection purposes) a quantity that expresses - on a common scale for all ionizing radiation - the approximate biological effectiveness of the absorbed dose.

Rad—The unit of absorbed dose equal to 100 ergs per gram, or 0.01 joule per kilogram (0.01 Gy) in any medium (see Absorbed Dose).

Radiation—The emission and propagation of energy through space or through a material medium in the form of waves (e.g., the emission and propagation of electromagnetic waves, or of sound and elastic waves). The term radiation or radiant energy, when unqualified, usually refers to electromagnetic radiation. Such radiation commonly is classified according to frequency, as microwaves, infrared, visible (light), ultraviolet, and x and gamma rays (see Photon.) and, by extension, corpuscular emission, such as alpha and beta radiation, neutrons, or rays of mixed or unknown type, as cosmic radiation.

Radiation, Annihilation—Photons produced when an electron and a positron unite and cease to exist. The annihilation of a positron-electron pair results in the production of two photons, each of 0.51 MeV energy.

Radiation, Background—See Background Radiation.

Radiation, Characteristic (Discrete)—Radiation originating from an excited atom after removal of an electron from an atom. The wavelength of the emitted radiation is specific, depending only on the element and particular energy levels involved.

Radiation, External—Radiation from a source outside the body.

Radiation, Internal—Radiation from a source within the body (as a result of deposition of radionuclides in body tissues).

Radiation, Ionizing—Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter (see Radiation).

Radiation, Monoenergetic—Radiation of a given type in which all particles or photons originate with and have the same energy.

Radiation, Scattered—Radiation which during its passage through a substance, has been deviated in direction. It may also have been modified by a decrease in energy.

Radiation, Secondary—A particle or ray that is produced when the primary radiation interacts with a material, and which has sufficient energy to produce its own ionization, such as bremsstrahlung or electrons knocked from atomic orbitals with enough energy to then produce ionization (see Delta Rays).

Radioactive Material—Material containing radioactive atoms.

Radioactivity—Spontaneous nuclear transformations that result in the formation of new elements. These transformations are accomplished by emission of alpha or beta particles from the nucleus or by the capture of an orbital electron. Each of these reactions may or may not be accompanied by a gamma photon.

Radioactivity, Artificial—Man-made radioactivity produced by particle bombardment or nuclear fission, as opposed to naturally occurring radioactivity.

Radioactivity, Induced—Radioactivity produced in a substance after bombardment with neutrons or other particles. The resulting activity is "natural radioactivity" if formed by nuclear reactions occurring in nature and "artificial radioactivity" if the reactions are caused by man.

Radioactivity, Natural—The property of radioactivity exhibited by more than 50 naturally occurring radionuclides.

Radioisotope—An unstable or radioactive isotope of an element that decays or disintegrates spontaneously, emitting radiation. Approximately 5,000 natural and artificial radioisotopes have been identified.

Radionuclide—Any radioactive isotope of any element.

Radiosensitivity—Relative susceptibility of cells, tissues, organs, organisms, or any living substance to the injurious action of radiation. Radiosensitivity and its antonym, radioresistance, are currently used in a comparative sense, rather than in an absolute one.

Reference Dose (RfD)—An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to non-threshold effects such as cancer.

Relative Biological Effectiveness (RBE)—The RBE is a factor used to compare the biological effectiveness of absorbed radiation doses (i.e., rad) due to different types of ionizing radiation. More specifically, it is the experimentally determined ratio of an absorbed dose of a radiation in question to the absorbed dose of a reference radiation (typically ^{60}Co gamma rays or 200 keV x rays) required to produce an identical biological effect in a particular experimental organism or tissue (see Quality Factor).

Rem—A unit of dose equivalent that is used in the regulatory, administrative, and engineering design aspects of radiation safety practice. The dose equivalent in rem is numerically equal to the absorbed dose in rad multiplied by the quality factor (1 rem is equal to 0.01 sievert).

Rep—Roentgen equivalent, physical: A former unit of ionizing radiation equivalent to the dosage of 93 ergs absorbed per gram of tissue (93 erg/gm=0.93 rad).

Reportable Quantity (RQ)—The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity—The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Roentgen (R)—A unit of exposure (in air) to ionizing radiation. It is the amount of x or gamma rays required to produce ions carrying 1 electrostatic unit of electrical charge in 1 cubic centimeter of dry air under standard conditions. Named after William Roentgen, a German scientist who discovered x rays in 1895.

Retrospective Study—A type of cohort study based on a group of persons known to have been exposed at some time in the past. Data are collected from routinely recorded events, up to the time the study is undertaken. Retrospective studies are limited to causal factors that can be ascertained from existing records and/or examining survivors of the cohort.

Self-Absorption—Absorption of radiation (emitted by radioactive atoms) by the material in which the atoms are located; in particular, the absorption of radiation within a sample being assayed.

Short-Term Exposure Limit (STEL)—The maximum concentration to which workers can be exposed for up to 15 min continually. No more than four excursions are allowed per day, and there must be at least 60 min between exposure periods. The daily TLV-TWA may not be exceeded.

SI Units—The International System of Units as defined by the General Conference of Weights and Measures in 1960. These units are generally based on the meter/kilogram/second units, with special quantities for radiation including the becquerel, gray, and sievert.

Sickness, Acute Radiation (Syndrome)—The complex symptoms and signs characterizing the condition resulting from excessive exposure of the whole body (or large part) to ionizing radiation. The earliest of these symptoms are nausea, fatigue, vomiting, and diarrhea, and may be followed by loss of hair (epilation), hemorrhage, inflammation of the mouth and throat, and general loss of energy. In severe cases, where the radiation dose is relatively high (over several hundred rad or several gray), death may occur within two to four weeks. Those who survive six weeks after exposure of a single high dose of radiation may generally be expected to recover.

Sievert (Sv)—The SI unit of any of the quantities expressed as dose equivalent. The dose equivalent in sieverts is equal to the absorbed dose, in gray, multiplied by the quality factor (1 sievert equals 100 rem).

Specific-activity—Radioactivity per unit mass of material containing a radionuclide, expressed, for example, as Ci/gram or Bq/gram.

Specific Energy—The actual energy per unit mass deposited per unit volume in a small target, such as the cell or cell nucleus, as the result of one or more energy-depositing events. This is a stochastic quantity as opposed to the average value over a large number of instance (i.e., the absorbed dose).

Standard Mortality Ratio (SMR)—A ratio of the observed number of deaths and the expected number of deaths in a specific standard population.

Stopping Power—The average rate of energy loss of a charged particle per unit thickness of a material or per unit mass of material traversed.

Surface-seeking Radionuclide—A bone-seeking internal emitter that is deposited and remains on the bone surface for a long period of time, although it may eventually diffuse into the bone mineral. This contrasts with a volume seeker, which deposits more uniformly throughout the bone volume.

Target Organ Toxicity—This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Target Theory (Hit Theory)—A theory explaining some biological effects of radiation on the basis that ionization, occurring in a discrete volume (the target) within the cell, directly causes a lesion which subsequently results in a physiological response to the damage at that location. One, two, or more "hits" (ionizing events within the target) may be necessary to elicit the response.

Teratogen—A chemical that causes birth defects.

Threshold Limit Value (TLV)—The maximum concentration of a substance to which most workers can be exposed without adverse effect. TLV is a term used exclusively by the ACGIH. Other terms used to express the same concept are the MAC (Maximum Allowable Concentration) and PEL (Permissible Exposure Limits).

Time-Weighted Average (TWA)—An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Toxic Dose (TD_{50})—A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Toxicokinetic—The study of the absorption, distribution and elimination of toxic compounds in the living organism.

Toxicosis —A diseased condition resulting from poisoning.

Transformation, Nuclear—The process by which a nuclide is transformed into a different nuclide by absorbing or emitting particulate or electromagnetic radiation.

Transition, Isomeric—The process by which a nuclide decays to an isomeric nuclide (i.e., one of the same mass number and atomic number) of lower quantum energy. Isomeric transitions (often abbreviated I.T.) proceed by gamma ray and/or internal conversion electron emission.

Tritium—The hydrogen isotope with one proton and two neutrons in the nucleus (Symbol: ^{3}H). It is radioactive and has a physical half-life of 12.3 years.

Unattached Fraction—That fraction of the radon daughters, usually ^{218}Po and ^{214}Po, which has not yet attached to a dust particle or to water vapor. As a free atom, it has a high probability of being exhaled and not retained within the lung. It is the attached fraction which is primarily retained.

Uncertainty Factor (UF)—A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

Units, Radiological—

Units	Equivalents
Becquerel* (Bq)	1 disintegration per second = $2.7x10^{-11}$ Ci
Curie (Ci)	$3.7x10^{10}$ disintegrations per second = $3.7x10^{10}$ Bq
Gray* (Gy)	1 J/kg = 100 rad
Rad (rad)	100 erg/g = 0.01 Gy
Rem (rem)	0.01 sievert
Sievert* (Sv)	100 rem

*International Units, designated (SI)

Working Level (WL)—Any combination of short-lived radon daughters in 1 liter of air that will result in the ultimate emission of $1.3x10^5$ MeV of potential alpha energy.

Working Level Month (WLM)—A unit of exposure to radon daughters corresponding to the product of the radon daughter concentration in Working Level (WL) and the exposure time in nominal months (1 nominal month = 170 hours). Inhalation of air with a concentration of 1 WL of radon daughters for 170 working hours results in an exposure of 1 WLM.

X rays—Penetrating electromagnetic radiations whose wave lengths are very much shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. X rays (called characteristic x rays) are also produced when an orbital electron falls from a high energy level to a low energy level.

Zero-Threshold Linear Hypothesis—The assumption that a dose-response curve derived from data in the high dose and high dose-rate ranges may be extrapolated through the low dose and low dose range to zero, implying that, theoretically, any amount of radiation will cause some damage.

APPENDIX A
ATSDR MINIMAL RISK LEVEL AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99–499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1–14 days), intermediate (15–364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as a hundredfold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agencywide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road, Mailstop E-29, Atlanta, Georgia 30333.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical name: Radioactive Cesium
CAS number: Multiple
Date: April 20, 2001
Profile status: Draft 3/Camera Ready
Route: [ ] Inhalation [ ] Oral [X] External
Duration: [X] Acute [ ] Intermediate [ ] Chronic
Species: Human

MRL: 4 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv (400 mrem)

References:

Schull WJ, Otake M and Yoshimaru H (1988). Effect on intelligence test score of prenatal exposure to ionizing radiation in Hiroshima and Nagasaki: A comparison of the T65DR and DS86 dosimetry systems.

Burt C. (1966). The genetic determination of differences in intelligence: A study of monozygotic twins reared together and apart. Brit. J. Psychol. 57 (1 & 2): pp. 137-153.

Experimental design:

Schull et al. (1988) study: Schull et al. (1988) evaluated the quantitative effect of exposure to ionizing radiation on the developing fetal and embryonic human brain. The end point measured was changes in intelligence test scores. The effects on individuals exposed *in utero* to the atomic bombing of Hiroshima and Nagasaki were based on the original PE86 samples (n=1,759; data on available intelligence testing) and a clinical sample (n=1,598). The original PE86 sample included virtually all prenatally exposed individuals who received tissue-absorbed doses of 0.50 Gy or more. There were many more individuals in the dose range 0–0.49 Gy in the PE86 sample than in the clinical sample. The clinical sample does not include children prenatally exposed at distances between 2,000–2,999 m in Hiroshima and Nagasaki. Children exposed at greater distances or not present in the city were selected as controls. In 1955–1956, Tanaka-B (emphasis on word-sense, arithmetic abilities, and the like, which were associated with the more subtle processing of visual clues than their simple recognition and depended more on connectedness) and the Koga (emphasis on perception of spatial relationships) intelligence tests were conducted in Nagasaki and the Koga test in Hiroshima.

Burt (1966) study: This study determined differences in intelligence in monozygotic twins reared together (n=95) and apart (n=53). All tests conducted in school consisted of (1) a group test of intelligence containing both non-verbal and verbal items, (2) an individual test (the London Revision of the Terman-Binet Scale) used primarily for standardization and for doubtful cases, and (3) a set of performance tests, based on the Pitner-Paterson tests and standardization. The methods and standard remained much the same throughout the study. Some of the reasons for separation of the twins were given as follows: death of the mother (n=9), unable to bring them up properly, mother's poor health (n=12), unmarried (n=6), and economic difficulties. The children were brought up by parents or foster parents (occupation ranged from unskilled to professional). IQ scores in the study group ranged from 66 to 137. The standard deviation of the group of separated monozygotic twins was reported at 15.3 as compared to 15.0 of ordinary siblings. Twins brought up in different environments were compared with those brought up in similar circumstances.

Effects noted in study and corresponding doses:

Schull et al. (1986) study: No evidence of radiation-related effect on intelligence was observed among individuals exposed within 0–7 weeks after fertilization or in the 26th or subsequent weeks. The highest risk of radiation damage to the embryonic and fetal brain occurs 8–15 weeks after fertilization under both dosimetric systems. The regression of intelligence score on estimated DS86 uterine absorbed dose is linear with dose, and the diminution in intelligence score is 21–29 points per Gy for the 8–15-week group and 10–26 points per Gy for the 16–25-week group. The results for 8–15 weeks applies regardless whether or not the mentally retarded individuals were included. The cumulative distribution of test scores suggested a progressive shift downwards in individual scores with increasing exposure. The mean IQ scores decrease significantly and systematically with uterine or fetal tissue dose within the 8–15 and 16–25-week groups.

In summary, analysis of intelligence test scores at 10–11 years of age of individuals exposed prenatally showed that:

- There is no evidence of a radiation-related effect on intelligence scores among those individuals exposed within 0–7 weeks of fertilization or in the 26th week of gestation and beyond;
- The cumulative distribution of test scores suggests a progressive shift downwards in intelligence scores with increasing exposure to ionizing radiation (dose-response relationship).
- The most sensitive group was the 8–15 weeks exposure group. The regression in intelligence scores was found to be linear, with 1 Gy dose resulting in a 21–29 point decline in intelligence scores.
- There was no indication of groups of individuals with differing sensitivities to radiation.

Burt (1966) study: The average intelligence of the twins measured on a conventional IQ scale (SD=15) was 97.8 for the separated monozygotes, 98.1 for monozygotes brought up together, 99.3 for the dizygotes as compared with 100.2 for the siblings, and 100.0 for the population as a whole. The difference of 0.3 IQ point between the separated and unseparated identical twins is considered a NOAEL for this study.

Dose endpoint used for MRL derivation:

[X] NOAEL [ ] LOAEL 0.3 IQ point reduction in twins, between those raised together and those raised apart.

Uncertainty factors (UF) used in MRL derivation:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability/sensitive population)

Was a conversion factor used from ppm in food or water to a mg/body weight dose?
If so, explain:

No.

If an inhalation study in animals, list conversion factors used in determining human equivalent dose:

Not applicable.

Was a conversion used from intermittent to continuous exposure?

No.

Other additional studies or pertinent information that lend support to this MRL:

Husen (1959) reported a study involving 269 pairs of Swedish monozygotic (identical) twins where the intrapair IQ difference was 4 IQ points for a combination of twins raised together and apart. This is somewhat lower than the value of 7 IQ points for identical twins raised apart, and just larger than the range of IQ scores for Washington, DC children repetitively tested (Jacobi and Glauberman 1995).

Supporting evidence for the acute MRL is provided by Jacobi and Glauberman (1995). Children in the 1st, 3rd, and 5th grades born in Washington, DC were tested, and average IQ levels of 94.2, 97.6, and 94.6 were reported. The range of 3.4 IQ points is considered to be a LOAEL for this study, which, if used for MRL derivation, would yield an MRL of 0.004 Sv (3.4 IQ points x 1 Sv/25 IQ points ÷ 30 [10 for use of a LOAEL and 3 for a sensitive population]).

Additional supporting evidence for the acute MRL is provided by Berger et al. 1997, in a case study of accidental radiation injury to the hand. A Mexican engineer suffered an accidental injury to the hand while repairing an x-ray spectrometer. The day after the accident, his symptoms included a tingling sensation and itching in the index and middle fingers. On days 4 and 7, a "pinching" sensation, swelling, and slight erythema were observed. By day 7, the tip of his index fingers was erythematous and a large blister developed with swelling on other fingers. On day 10, examination by a physician showed that the lesions had worsened and the fingers and palms were discolored. On day 10, he was admitted to the hospital where hyperbaric oxygen therapy was administered without success. One month after the accident, the patient entered the hospital again with pain, discoloration, and desquamation of his hand. Clinical examination showed decreased circulation in the entire hand, most notably in the index and middle finger. Total white blood count decreased to 3,000/µL (normal range 4,300–10,800/µL). Cytogenic studies of peripheral blood lymphocytes revealed four dicentrics, two rings, and eight chromosomal fragments in the 300 metaphases studied. The estimated whole body dose was reported to be 0.382 Gy (38.2 rad). This dose is a potential LOAEL for acute ionizing radiation and would yield an MRL of 0.004 Sv (0.38 Sv ÷100 [10 for use of LOAEL and 10 for sensitive human population]).

The NRC set a radiation exposure limit of 0.5 rem (50 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks ATSDR believes that the conservative acute MRL of 4 mSv is consistent with the NRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

Calculations

Given: 0.3 IQ point is a NOAEL. A 1 Sv dose results in a 25 IQ point reduction (range = 21–29 points; mean=25) and provides a conversion factor from IQ prediction to radiation dose. Assume that the radiation dose and the subsequent reduction in IQ is a linear relationship.

MRL = NOAEL x CF ÷ UF
MRL = 0.3 x 1/25 ÷ 3
MRL = 0.004 Sv = 4 mSv (400 mrem)

Agency Contact (Chemical Manager):

Malcolm Williams, Ph.D.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical name: Radioactive Cesium
CAS number: Multiple
Date: April 20, 2001
Profile status: Draft 3/Camera Ready
Route: [ ] Inhalation [ ] Oral [X] External
Duration: [ ] Acute [ ] Intermediate [X] Chronic
Species: Human

MRL: 1 [] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv/year (100 mrem/year)

Reference: BEIR V. 1990. Health effects of exposure to low levels of ionizing radiation. Committee on the Biological Effects of Ionizing Radiations, National Research Council. National Academy Press. Washington, DC.

Experimental design: Not applicable

Effects noted in study and corresponding doses:

No individual studies were identified that could be used to base a chronic-duration external exposure MRL that did not result in a cancer-producing end point. However, two sources of information were identified that did provide doses of ionizing radiation that have not been reported to be associated with detrimental effects (NOAELs). These sources provide estimates of background levels of primarily natural sources of ionizing radiation that have not been implicated in producing cancerous or non-cancerous toxicological endpoints. BEIR V states that the average annual effective dose to the U.S. population is 3.6 mSv/year. A total annual effective dose equivalent of 3.6 mSv (360 mrem)/year to members of the U.S. population is obtained mainly by naturally occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. The largest contribution (82%) is from natural sources, two-thirds of which is from naturally occurring radon and its decay products. Specific sources of this radiation are demonstrated in Table A-1.

The annual dose of 3.6 mSv per year has not been associated with adverse health effects or increases in the incidences of any type of cancers in humans or other animals.

Dose endpoint used for MRL derivation: 3.6 mSv/year

[X] NOAEL [ ] LOAEL 3.6 mSv/year

Uncertainty factors (UF) used in MRL derivation:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability)

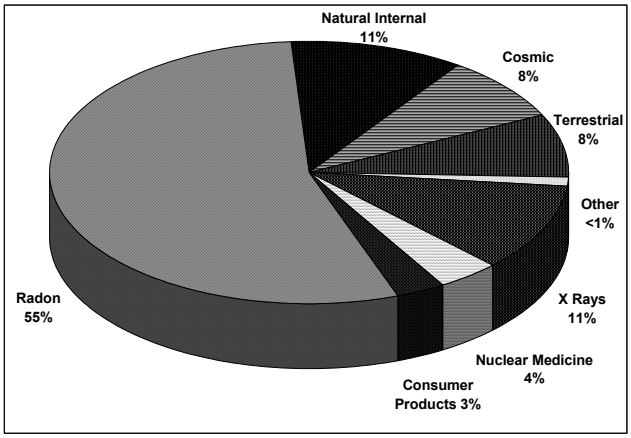
Was a conversion factor used from ppm in food or water to a mg/body weight dose?
If so, explain:

No.

APPENDIX A

Table A-1. Average Annual Effective Dose Equivalent of Ionizing Radiation to a Member of the U.S. Population[a]

Source	Effective Dose Equivalent	
	mSv	Percent of Total Dose
Natural		
Radon[b]	2.0	55
Cosmic	0.27	8.0
Terrestrial	0.28	8.0
Internal	0.39	11
Total Natural	3.0	82
Artificial		
Medical		
X-ray	0.39	11
Nuclear	0.14	4.0
Consumer Products	0.10	3.0
Other		
Occupational	<0.01	<0.3
Nuclear Fuel Cycle	<0.01	<0.03
Fallout	<0.01	<0.03
Miscellaneous[c]	<0.01	<0.03
Total Artificial	0.63	18
Total Natural and Artificial	3.6	100



[a]adapted from BEIR V, Table 1-3 , page 18.
[b]Dose equivalent to bronci from radon daughter products
[c]DOE facilities, smelter, transportation, etc.

If an inhalation study in animals, list conversion factors used in determining human equivalent dose:

Not applicable.

Was a conversion used from intermittent to continuous exposure?

No.

Other additional studies or pertinent information that lend support to this MRL:

ICRP has developed recommended dose limits for occupational and public exposure to ionizing radiation sources. The ICRP recommends limiting public exposure to 1 mSv/year (100 mrem/year), but does note that values at high altitudes above sea level and in some geological areas can sometimes be twice that value ($2 mSv). In Annex C of ICRP 60, the commission provides projections of risk from the high dose, high dose rate Japanese atomic bomb survivor data and a relatively low dose, low dose rate Swedish population (considering a dose and dose rate effectiveness factor of 2) that predict a potentially small increase in age-specific human mortality rate with increasing radiation dose from 1 mSv to 5 mSv. This increase may not be able to be detected in a typical population. ICRP states that the value of 1 mSv/year was chosen over the 5 mSv value because 5 mSv/year (500 mrem/year) could result in a small increase in age specific mortality rate, and 1 mSv/year (100 mrem/year) is typical of the annual effective dose from background, less radon (ICRP 1991). The 1 mSv estimate may underestimate the annual exposure to external sources of ionizing radiation to the U.S. population, as it does not include radiation from radon. Conversely, the 5 mSv estimate may be high, in that this larger dose could potentially result in a small mortality increase. The most useful estimate appears to be the BEIR V estimate of 3.6 mSv as a nationwide average that accounts for an annual exposure to radon, is specific to the U.S. population, has not been associated with increases mortality, and it falls short of the 5 mSv value potentially associated with small increases in human mortality.

Calculations

$MRL = NOAEL_{(ADJ)} \div UF$
MRL = 3.6 mSv/year ÷ 3
MRL = 1.20 mSv/year
MRL = 1.0 mSv/year = 100 mrem/year above background

Agency Contact (Chemical Manager):

Malcolm Williams, Ph.D.

APPENDIX B

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Relevance to Public Health

This chapter provides an overview of the nature, manufacture, uses, general population exposures, and health effects of the substance under review. This overview is followed by a discussion of the most critical health effects. The discussion of health effects is based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?

2. What effects observed in animals are likely to be of concern to humans?

3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The chapter covers end points in the same order they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this chapter. If data are located in the scientific literature, a table of genotoxicity information is included.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. Minimal risk levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Chapter 3 Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, we have derived minimal risk levels (MRLs) for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic). These MRLs are not meant to support regulatory action; but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2, "Relevance to Public Health," contains basic information known about the substance. Other sections such as Chapter 3 Section 3.9, "Interactions with Other Substances," and Section 3.10, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses for lifetime exposure (RfDs).

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables (3-1, 3-2, and 3-3) and figures (3-1 and 3-2) are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, minimal risk levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse- Effect Levels (NOAELs), Lowest-Observed-Adverse-Effect Levels (LOAELs), or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples of LSE Table 3-1 and Figure 3-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See LSE Table 3-1

(1) Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exists, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 3-1, 3-2, and 3-3, respectively). LSE figures are limited to the inhalation (LSE Figure 3-1) and oral (LSE Figure 3-2) routes. Not all substances will have data on each route of exposure and will not therefore have all five of the tables and figures. In the case of radionuclide profiles, there are separate LSE tables, by route of exposure, for toxicity of stable isotopes and radioactive isotopes; for radioactive isotopes, there may be an LSE table for a fourth route, external exposure.

(2) Exposure Period Three exposure periods - acute (less than 15 days), intermediate (15–364 days), and chronic (365 days or more) are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.

(3) Health Effect The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table (see key number 18).

(4) Key to Figure Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the 2 "18r" data points in Figure 3-1).

(5) Species The test species, whether animal or human, are identified in this column. Chapter 2, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 3.4, "Toxicokinetics," contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(6) Exposure Frequency/Duration The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to 1,1,2,2-tetrachloroethane via inhalation for 6 hours per day, 5 days per week, for 3 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper, i.e., Nitschke et al. 1981.

(7) System This column further defines the systemic effects. These systems include: respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, 1 systemic effect (respiratory) was investigated.

(8) NOAEL A No-Observed-Adverse-Effect Level (NOAEL) is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system which was used to derive an intermediate exposure, inhalation MRL of 0.005 ppm (see footnote "b").

(9) LOAEL A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.

(10) Reference The complete reference citation is given in Chapter 9 of the profile.

(11) CEL A Cancer Effect Level (CEL) is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiologic studies. CELs are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses not causing measurable cancer increases.

(12) Footnotes Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "b" indicates the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See Figure 3-1

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure concentrations for particular exposure periods.

(13) Exposure Period The same exposure periods appear as in the LSE table. In this example, health effects observed within the intermediate and chronic exposure periods are illustrated.

(14) Health Effect These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.

(15) Levels of Exposure concentrations or doses for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis. Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16) NOAEL In this example, 18r NOAEL is the critical end point for which an intermediate inhalation exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates to a NOAEL for the test species-rat. The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17) CEL Key number 38r is 1 of 3 studies for which Cancer Effect Levels were derived. The diamond symbol refers to a Cancer Effect Level for the test species-mouse. The number 38 corresponds to the entry in the LSE table.

(18) Estimated Upper-Bound Human Cancer Risk Levels This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

(19) Key to LSE Figure The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 6

Table 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation

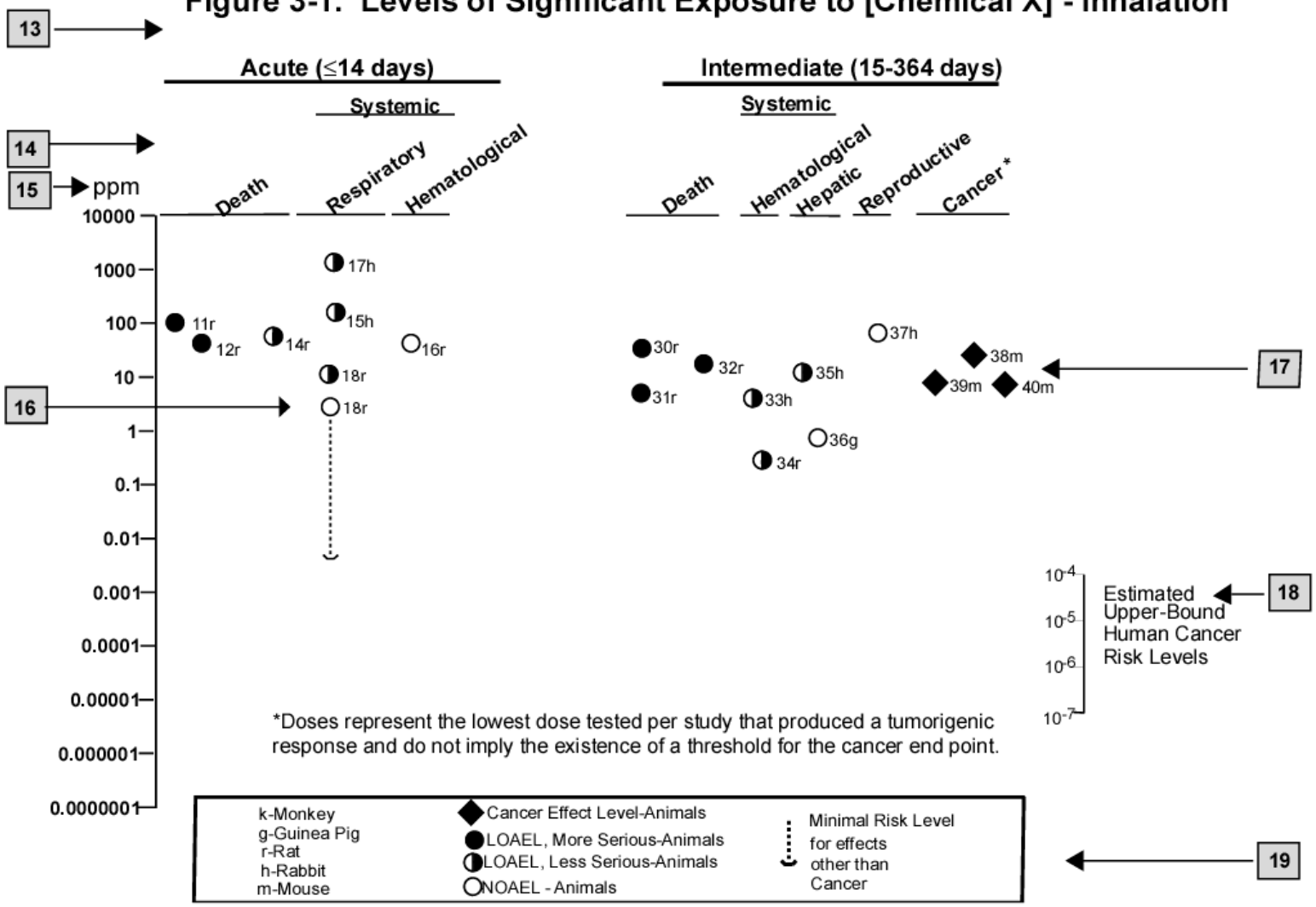
Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 6 INTERMEDIATE EXPOSURE							
	5	6	7	8	9		10
3 6 Systemic	9	9	9	9	9		9
4 6 18	Rat	13 wk 5 d/wk 6 hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
CHRONIC EXPOSURE							
					11		
Cancer					9		
38	Rat	18 mo 5 d/wk 7 hr/d			20	(CEL, multiple organs)	Wong et al. 1982
39	Rat	89–104 wk 5 d/wk 6 hr/d			10	(CEL, lung tumors, nasal tumors)	NTP 1982
40	Mouse	79–103 wk 5 d/wk 6 hr/d			10	(CEL, lung tumors, hemangiosarcomas)	NTP 1982

[a] The number corresponds to entries in Figure 3-1.

12 6 [b] Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5 x 10^{-3} ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

SAMPLE

Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation



APPENDIX C

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADI	Acceptable Daily Intake
ADME	Absorption, Distribution, Metabolism, and Excretion
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAT	Best Available Technology
BCF	bioconcentration factor
BEI	Biological Exposure Index
BSC	Board of Scientific Counselors
C	Centigrade
CAA	Clean Air Act
CAG	Cancer Assessment Group of the U.S. Environmental Protection Agency
CAS	Chemical Abstract Services
CDC	Centers for Disease Control and Prevention
CEL	Cancer Effect Level
CELDS	Computer-Environmental Legislative Data System
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
Ci	curie
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CNS	central nervous system
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
d	day
Derm	dermal
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNA	deoxyribonucleic acid
DOD	Department of Defense
DOE	Department of Energy
DOL	Department of Labor
DOT	Department of Transportation
DOT/UN/ NA/IMCO	Department of Transportation/United Nations/ North America/International Maritime Dangerous Goods Code
DWEL	Drinking Water Exposure Level
ECD	electron capture detection
ECG/EKG	electrocardiogram

EEG	electroencephalogram
EEGL	Emergency Exposure Guidance Level
EPA	Environmental Protection Agency
F	Fahrenheit
F_1	first-filial generation
FAO	Food and Agricultural Organization of the United Nations
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FPD	flame photometric detection
fpm	feet per minute
ft	foot
FR	*Federal Register*
g	gram
GC	gas chromatography
Gd	gestational day
gen	generation
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
hr	hour
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IDLH	Immediately Dangerous to Life and Health
IARC	International Agency for Research on Cancer
ILO	International Labor Organization
in	inch
IRIS	Integrated Risk Information System
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{Lo}	lethal concentration, low
LC_{50}	lethal concentration, 50% kill
LD_{Lo}	lethal dose, low
LD_{50}	lethal dose, 50% kill
LT_{50}	lethal time, 50% kill
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
m	meter
MA	*trans,trans*-muconic acid
MAL	Maximum Allowable Level
mCi	millicurie
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
mg	milligram
min	minute
mL	milliliter

mm	millimeter
mm Hg	millimeters of mercury
mmol	millimole
mo	month
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAAQS	National Ambient Air Quality Standard
NAS	National Academy of Science
NATICH	National Air Toxics Information Clearinghouse
NATO	North Atlantic Treaty Organization
NCE	normochromatic erythrocytes
NCI	National Cancer Institute
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NFPA	National Fire Protection Association
ng	nanogram
NLM	National Library of Medicine
nm	nanometer
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPD	nitrogen phosphorus detection
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NR	not reported
NRC	National Research Council; Nuclear Regulatory Commission
NS	not specified
NSPS	New Source Performance Standards
NTIS	National Technical Information Service
NTP	National Toxicology Program
ODW	Office of Drinking Water, EPA
OERR	Office of Emergency and Remedial Response, EPA
OHM/TADS	Oil and Hazardous Materials/Technical Assistance Data System
OPP	Office of Pesticide Programs, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OPPT	Office of Pollution Prevention and Toxics, EPA
OSHA	Occupational Safety and Health Administration
OSW	Office of Solid Waste, EPA
OTS	Office of Toxic Substances
OW	Office of Water
OWRS	Office of Water Regulations and Standards, EPA
PAH	Polycyclic Aromatic Hydrocarbon
PBPD	Physiologically Based Pharmacodynamic
PBPK	Physiologically Based Pharmacokinetic
PCE	polychromatic erythrocytes
PEL	permissible exposure limit
PID	photo ionization detector

pg	picogram
pmol	picomole
PHS	Public Health Service
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	Pretreatment Standards for New Sources
REL	recommended exposure level/limit
RfC	Reference Concentration
RfD	Reference Dose
RNA	ribonucleic acid
RTECS	Registry of Toxic Effects of Chemical Substances
RQ	Reportable Quantity
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
sec	second
SIC	Standard Industrial Classification
SIM	selected ion monitoring
SMCL	Secondary Maximum Contaminant Level
SMR	standard mortality ratio
SNARL	Suggested No Adverse Response Level
SPEGL	Short-Term Public Emergency Guidance Level
STEL	short term exposure limit
STORET	Storage and Retrieval
TD_{50}	toxic dose, 50% specific toxic effect
TLV	threshold limit value
TOC	Total Organic Compound
TPQ	Threshold Planning Quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TRI	Toxics Release Inventory
TWA	time-weighted average
U.S.	United States
UF	uncertainty factor
VOC	Volatile Organic Compound
yr	year
WHO	World Health Organization
wk	week

>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
γ	gamma
δ	delta
μm	micrometer

µg	microgram
q_1^*	cancer slope factor
–	negative
+	positive
(+)	weakly positive result
(–)	weakly negative result

APPENDIX D

OVERVIEW OF BASIC RADIATION PHYSICS, CHEMISTRY, AND BIOLOGY

Understanding the basic concepts in radiation physics, chemistry, and biology is important to the evaluation and interpretation of radiation-induced adverse health effects and to the derivation of radiation protection principles. This appendix presents a brief overview of the areas of radiation physics, chemistry, and biology and is based to a large extent on the reviews of Mettler and Moseley (1985), Hobbs and McClellan (1986), Eichholz (1982), Hendee (1973), Cember (1996), and Early et al. (1979).

D.1 RADIONUCLIDES AND RADIOACTIVITY

The substances we call elements are composed of atoms. Atoms in turn are made up of neutrons, protons and electrons: neutrons and protons in the nucleus and electrons in a cloud of orbits around the nucleus. Nuclide is the general term referring to any nucleus along with its orbital electrons. The nuclide is characterized by the composition of its nucleus and hence by the number of protons and neutrons in the nucleus. All atoms of an element have the same number of protons (this is given by the atomic number) but may have different numbers of neutrons (this is reflected by the atomic mass numbers or atomic weight of the element). Atoms with different atomic mass but the same atomic numbers are referred to as isotopes of an element.

The numerical combination of protons and neutrons in most nuclides is such that the nucleus is quantum mechanically stable and the atom is said to be stable, i.e., not radioactive; however, if there are too few or too many neutrons, the nucleus is unstable and the atom is said to be radioactive. Unstable nuclides undergo radioactive transformation, a process in which a neutron or proton converts into the other and a beta particle is emitted, or else an alpha particle is emitted. Each type of decay is typically accompanied by the emission of gamma rays. These unstable atoms are called radionuclides; their emissions are called ionizing radiation; and the whole property is called radioactivity. Transformation or decay results in the formation of new nuclides some of which may themselves be radionuclides, while others are stable nuclides. This series of transformations is called the decay chain of the radionuclide. The first radionuclide in the chain is called the parent; the subsequent products of the transformation are called progeny, daughters, or decay products.

In general there are two classifications of radioactivity and radionuclides: natural and artificial (man-made). Naturally-occurring radioactive material (NORM) exists in nature and no additional energy is necessary to place them in an unstable state. Natural radioactivity is the property of some naturally occurring, usually heavy elements, that are heavier than lead. Radionuclides, such as radium and uranium, primarily emit alpha particles. Some lighter elements such as carbon-14 and tritium (hydrogen-3) primarily emit beta particles as they transform to a more stable atom. Natural radioactive atoms heavier than lead cannot attain a stable nucleus heavier than lead. Everyone is exposed to background radiation from naturally-occurring radionuclides throughout life. This background radiation is the major source of radiation exposure to man and arises from several sources. The natural background exposures are frequently used as a standard of comparison for exposures to various artificial sources of ionizing radiation.

Artificial radioactive atoms are produced either as a by-product of fission of uranium or plutonium atoms in a nuclear reactor or by bombarding stable atoms with particles, such as neutrons or protons, directed at the stable atoms with high velocity. These artificially produced radioactive elements usually decay by emission of particles, such as positive or negative beta particles and one or more high energy photons (gamma rays). Unstable (radioactive) atoms of any element can be produced.

Both naturally occurring and artificial radioisotopes find application in medicine, industrial products, and consumer products. Some specific radioisotopes, called fall-out, are still found in the environment as a result of nuclear weapons use or testing.

D.2 RADIOACTIVE DECAY

D.2.1 Principles of Radioactive Decay

The stability of an atom is the result of the balance of the forces of the various components of the nucleus. An atom that is unstable (radionuclide) will release energy (decay) in various ways and transform to stable atoms or to other radioactive species called daughters, often with the release of ionizing radiation. If there are either too many or too few neutrons for a given number of protons, the resulting nucleus may undergo transformation. For some elements, a chain of daughter decay products may be produced until stable atoms are formed. Radionuclides can be characterized by the type and energy of the radiation emitted, the rate of decay, and the mode of decay. The mode of decay indicates how a parent compound undergoes transformation. Radiations considered here are primarily of nuclear origin, i.e., they arise from nuclear excitation, usually caused by the capture of charged or uncharged nucleons by a nucleus, or by the radioactive decay or transformation of an unstable nuclide. The type of radiation may be categorized as charged or uncharged particles, protons, and fission products) or electromagnetic radiation (gamma rays and x rays). Table D-1 summarizes the basic characteristics of the more common types of radiation encountered.

D.2.2 Half-Life and Activity

For any given radionuclide, the rate of decay is a first-order process that is constant, regardless of the radioactive atoms present and is characteristic for each radionuclide. The process of decay is a series of random events; temperature, pressure, or chemical combinations do not effect the rate of decay. While it may not be possible to predict exactly which atom is going to undergo transformation at any given time, it is possible to predict, on average, the fraction of the radioactive atoms that will transform during any interval of time.

The *activity* is a measure of the quantity of radioactive material. For these radioactive materials it is customary to describe the activity as the number of disintegrations (transformations) per unit time. The unit of activity is the curie (Ci), which was originally related to the activity of one gram of radium, but is now defined as that quantity of radioactive material in which there are:

> 1 curie (Ci) = 3.7×10^{10} disintegrations (transformations)/second (dps) or 2.22×10^{12} disintegrations (transformations)/minute (dpm).

The SI unit of activity is the becquerel (Bq); 1 Bq = that quantity of radioactive material in which there is 1 transformation/second. Since activity is proportional to the number of atoms of the radioactive material, the quantity of any radioactive material is usually expressed in curies, regardless of its purity or concentration. The transformation of radioactive nuclei is a random process, and the number of transformations is directly proportional to the number of radioactive atoms present. For any pure radioactive substance, the rate of decay is usually described by its radiological half-life, T_R, i.e., the time it takes for a specified source material to decay to half its initial activity. The specific activity is an indirect measure of the rate of decay, and is defined as the activity per unit mass or per unit volume. The higher the specific activity of a radioisotope, the faster it is decaying.

The activity of a radionuclide at time t may be calculated by:

$$A = A_o e^{-0.693t/Trad}$$

where A is the activity in dps or curies or becquerels, A_o is the activity at time zero, t is the time at which measured, and T_{rad} is the radiological half-life of the radionuclide (T_{rad} and t must be in the same units of time). The time when the activity of a sample of radioactivity becomes one-half its original value is the radioactive half-life and is expressed in any suitable unit of time.

Table D-1. Characteristics of Nuclear Radiations

Radiation	Rest mass[a]	Charge	Typical energy range	Path length[b]		Comments
				Air	Solid	
Alpha (α)	4.00 amu	+2	4–10 MeV	5–10 cm	25–80 µm	Identical to ionized He nucleus
Negatron (β^-)	$5.48x10^{-4}$ amu; 0.51 MeV	–1	0–4 Mev	0–10 m	0–1 cm	Identical to electron
Positron (β^+)	$5.48x10^{-4}$ amu; 0.51 Mev	+1	0-4 Mev	0–10 m	0–1 cm	Identical to electron except for sign of charge
Neutron	1.0086 amu; 939.55 MeV	0	0–15 MeV	b	b	Free half-life: 16 min
X ray (e.m. photon)	–	0	5 keV–100 keV	b	b	Photon from transition of an electron between atomic orbits
Gamma (p) (e.m. photon)	–	0	10 keV–3 MeV	b	b	Photon from nuclear transformation

[a] The rest mass (in amu) has an energy equivalent in MeV that is obtained using the equation $E=mc^2$, where 1 amu = 932 MeV.
[b] Path lengths are not applicable to x- and gamma rays since their intensities decrease exponentially; path lengths in solid tissue are variable, depending on particle energy, electron density of material, and other factors.

amu = atomic mass unit; e.m. = electromagnetic; MeV = MegaElectron Volts

The specific activity is a measure of activity, and is fined as the activity per unit mass or per unit volume. This activity is usually expressed in curies per gram and may be calculated by

$$\text{curies/gram} = 1.3x10^8 / (T_{rad}) \text{ (atomic weight)} \quad \text{or}$$

$$[3.577 \times 10^5 \times \text{mass(g)}] / [T_{rad} \times \text{atomic weight}]$$

where T_{rad} is the radiological half-life in days.

In the case of radioactive materials contained in living organisms, an additional consideration is made for the reduction in observed activity due to regular processes of elimination of the respective chemical or biochemical substance from the organism. This introduces a rate constant called the biological half-life (T_{biol}) which is the time required for biological processes to eliminate one-half of the activity. This time is virtually the same for both stable and radioactive isotopes of any given element.

Under such conditions the time required for a radioactive element to be halved as a result of the combined action of radioactive decay and biological elimination is the effective clearance half-time:

$$T_{eff} = (T_{biol} \times T_{rad}) / (T_{biol} + T_{rad}).$$

Table D-2 presents representative effective half-lives of particular interest.

Table D-2. Half-Lives of Some Radionuclides in Adult Body Organs

		Half-life[a]		
Radionuclide	Critical organ	Physical	Biological	Effective
Uranium-238	Kidney	4,460,000,000 y	4 d	4 d
Hydrogen-3[b] (Tritium)	Whole body	12.3 y	10 d	10 d
Iodine-131	Thyroid	8 d	80 d	7.3 d
Strontium-90	Bone	28 y	50 y	18 y
Plutonium-239	Bone surface	24,400 y	50 y	50 y
	Lung	24,400 y	500 d	500 d
Cobalt-60	Whole body	5.3 y	99.5 d	95 d
Iron-55	Spleen	2.7 y	600 d	388 d
Iron-59	Spleen	45.1 d	600 d	42 d
Manganese-54	Liver	303 d	25 d	23 d
Cesium-137	Whole body	30 y	70 d	70 d

[a]d = days, y = years
[b]Mixed in body water as tritiated water

D.2.3 Interaction of Radiation with Matter

Both ionizing and nonionizing radiation will interact with materials; that is, radiation will lose kinetic energy to any solid, liquid or gas through which it passes by a variety of mechanisms. The transfer of energy to a medium by either electromagnetic or particulate radiation may be sufficient to cause formation of ions. This process is called ionization. Compared to other types of radiation that may be absorbed, such as ultraviolet radiation, ionizing radiation deposits a relatively large amount of energy into a small volume.

The method by which incident radiation interacts with the medium to cause ionization may be direct or indirect. Electromagnetic radiations (x rays and gamma photons) are indirectly ionizing; that is, they give up their energy in various interactions with cellular molecules, and the energy is then utilized to produce a fast-moving charged particle such as an electron. It is the electron that then may react with a target molecule. This particle is called a “primary ionizing particle. Charged particles, in contrast, strike the tissue or medium and directly react with target molecules, such as oxygen or water. These particulate radiations are directly ionizing radiations. Examples of directly ionizing particles include alpha and beta particles. Indirectly ionizing radiations are always more penetrating than directly ionizing particulate radiations.

Mass, charge, and velocity of a particle all affect the rate at which ionization occurs. The higher the charge of the particle and the lower the velocity, the greater the propensity to cause ionization. Heavy, highly charged particles, such as alpha particles, lose energy rapidly with distance and, therefore, do not penetrate deeply. The result of these interaction processes is a gradual slowing down of any incident particle until it is brought to rest or "stopped" at the end of its range.

D.2.4 Characteristics of Emitted Radiation

D.2.4.1 Alpha Emission. In alpha emission, an alpha particle consisting of two protons and two neutrons is emitted with a resulting decrease in the atomic mass number by four and reduction of the atomic number of two, thereby changing the parent to a different element. The alpha particle is identical to a helium nucleus consisting of two neutrons and two protons. It results from the radioactive decay of some heavy elements such as uranium, plutonium, radium, thorium, and radon. All alpha particles emitted by a given radioisotope have the same energy. Most of the alpha particles that are likely to be found have energies in the range of about 4 to 8 MeV, depending on the isotope from which they came.

The alpha particle has an electrical charge of +2. Because of this double positive charge and their size, alpha particles have great ionizing power and, thus, lose their kinetic energy quickly. This results in very little penetrating power. In fact, an alpha particle cannot penetrate a sheet of paper. The range of an alpha particle (the distance the charged particle travels from the point of origin to its resting point) is about 4 cm in air, which decreases considerably to a few micrometers in tissue. These properties cause alpha emitters to be hazardous only if there is internal contamination (i.e., if the radionuclide is inside the body).

D.2.4.2 Beta Emission. A beta particle (&) is a high-velocity electron ejected from a disintegrating nucleus. The particle may be either a negatively charged electron, termed a negatron (&-) or a positively charged electron, termed a positron (&+). Although the precise definition of "beta emission" refers to both &- and &+, common usage of the term generally applies only to the negative particle, as distinguished from the positron emission, which refers to the &+ particle.

D.2.4.2.1 Beta Negative Emission. Beta particle (&-) emission is another process by which a radionuclide, with a neutron excess achieves stability. Beta particle emission decreases the number of neutrons by one and increases the number of protons by one, while the atomic mass number remains unchanged.[1] This transformation results in the formation of a different element. The energy spectrum of beta particle emission ranges from a certain maximum down to zero with the mean energy of the spectrum being about one-third of the maximum. The range in tissue is much less. Beta negative emitting radionuclides can cause injury to the skin and superficial body tissues, but mostly present an internal contamination hazard.

D.2.4.2.2 Positron Emission. In cases in which there are too many protons in the nucleus, positron emission may occur. In this case a proton may be thought of as being converted into a neutron, and a positron (&+) is emitted.[1] This increases the number of neutrons by one, decreases the number of protons by one, and again leaves the atomic mass number unchanged. The gamma radiation resulting from the annihilation (see glossary) of the positron makes all positron emitting isotopes more of an external radiation hazard than pure & emitters of equal energy.

[1]Neutrinos also accompany negative beta particles and positron emissions.

D.2.4.2.3 Gamma Emission. Radioactive decay by alpha, beta, or positron emission, or electron capture often leaves some of the energy resulting from these changes in the nucleus. As a result, the nucleus is raised to an excited level. None of these excited nuclei can remain in this high-energy state. Nuclei release this energy returning to ground state or to the lowest possible stable energy level. The energy released is in the form of gamma radiation (high energy photons) and has an energy equal to the change in the energy state of the nucleus. Gamma and x rays behave similarly but differ in their origin; gamma emissions originate in the nucleus while x rays originate in the orbital electron structure or from rapidly changing the velocity of an electron (e.g., as occurs when shielding high energy beta particles or stopping the electron beam in an x ray tube).

D.3 ESTIMATION OF ENERGY DEPOSITION IN HUMAN TISSUES

Two forms of potential radiation exposures can result: internal and external. The term exposure denotes physical interaction of the radiation emitted from the radioactive material with cells and tissues of the human body. An exposure can be "acute" or "chronic" depending on how long an individual or organ is exposed to the radiation. Internal exposures occur when radionuclides, which have entered the body (e.g., through the inhalation, ingestion, or dermal pathways), undergo radioactive decay resulting in the deposition of energy to internal organs. External exposures occur when radiation enters the body directly from sources located outside the body, such as radiation emitters from radionuclides on ground surfaces, dissolved in water, or dispersed in the air. In general, external exposures are from material emitting gamma radiation, which readily penetrate the skin and internal organs. Beta and alpha radiation from external sources are far less penetrating and deposit their energy primarily on the skin's outer layer. Consequently, their contribution to the absorbed dose of the total body dose, compared to that deposited by gamma rays, may be negligible.

Characterizing the radiation dose to persons as a result of exposure to radiation is a complex issue. It is difficult to: (1) measure internally the amount of energy actually transferred to an organic material and to correlate any observed effects with this energy deposition; and (2) account for and predict secondary processes, such as collision effects or biologically triggered effects, that are an indirect consequence of the primary interaction event.

D.3.1 Dose/Exposure Units

D.3.1.1 Roentgen. The roentgen (R) is a unit of x or gamma-ray exposure and is a measured by the amount of ionization caused in air by gamma or x radiation. One roentgen produces 2.58×10^{-4} coulomb per kilogram of air. In the case of gamma radiation, over the commonly encountered range of photon energy, the energy deposition in tissue for a dose of 1 R is about 0.0096 joules (J) /kg of tissue.

D.3.1.2 Absorbed Dose and Absorbed Dose Rate. The absorbed dose is defined as the energy imparted by the incident radiation to a unit mass of the tissue or organ. The unit of absorbed dose is the rad; 1 rad = 100 erg/gram = 0.01 J/kg in any medium. An exposure of 1 R results in a dose to soft tissue of approximately 0.01 J/kg. The SI unit is the gray which is equivalent to 100 rad or 1 J/kg. Internal and external exposures from radiation sources are not usually instantaneous but are distributed over extended periods of time. The resulting rate of change of the absorbed dose to a small volume of mass is referred to as the absorbed dose rate in units of rad/unit time.

D.3.1.3 Working Levels and Working Level Months. Working level (WL) is a measure of the atmospheric concentration of radon and its short-lived progeny. One WL is defined as any combination of short-lived radon daughters (through polonium-214), per liter of air, that will result in the emission of 1.3×10^5 MeV of alpha energy. An activity concentration of 100 pCi radon-222/L of air, in equilibrium with its daughters, corresponds approximately to a potential alpha-energy concentration of 1 WL. The WL unit can also be used for thoron daughters. In this case, 1.3×10^5 MeV of alpha energy (1 WL) is

released by the thoron daughters in equilibrium with 7.5 pCi thoron/L. The potential alpha energy exposure of miners is commonly expressed in the unit Working Level Month (WLM). One WLM corresponds to exposure to a concentration of 1 WL for the reference period of 170 hours, or more generally

WLM = concentration (WL) x exposure time (months) (one "month" = 170 working hours).

D.3.2 Dosimetry Models

Dosimetry models are used to estimate the dose from internally deposited to radioactive substances. The models for internal dosimetry consider the amount of radionuclides entering the body, the factors affecting their movement or transport through the body, distribution and retention of radionuclides in the body, and the energy deposited in organs and tissues from the radiation that is emitted during spontaneous decay processes. The dose pattern for radioactive materials in the body may be strongly influenced by the route of entry of the material. For industrial workers, inhalation of radioactive particles with pulmonary deposition and puncture wounds with subcutaneous deposition have been the most frequent. The general population has been exposed via ingestion and inhalation of low levels of naturally occurring radionuclides as well as radionuclides from nuclear weapons testing.

The models for external dosimetry consider only the photon doses to organs of individuals who are immersed in air or are exposed to a contaminated object.

D.3.2.1 Ingestion. Ingestion of radioactive materials is most likely to occur from contaminated foodstuffs or water or eventual ingestion of inhaled compounds initially deposited in the lung. Ingestion of radioactive material may result in toxic effects as a result of either absorption of the radionuclide or irradiation of the gastrointestinal tract during passage through the tract, or a combination of both. The fraction of a radioactive material absorbed from the gastrointestinal tract is variable, depending on the specific element, the physical and chemical form of the material ingested, and the diet, as well as some other metabolic and physiological factors. The absorption of some elements is influenced by age, usually with higher absorption in the very young.

D.3.2.2 Inhalation. The inhalation route of exposure has long been recognized as being a major portal of entry for both nonradioactive and radioactive materials. The deposition of particles within the lung is largely dependent upon the size of the particles being inhaled. After the particle is deposited, the retention will depend upon the physical and chemical properties of the dust and the physiological status of the lung. The retention of the particle in the lung depends on the location of deposition, in addition to the physical and chemical properties of the particles. The converse of pulmonary retention is pulmonary clearance. There are three distinct mechanisms of clearance which operate simultaneously. Ciliary clearance acts only in the upper respiratory tract. The second and third mechanisms act mainly in the deep respiratory tract. These are phagocytosis and absorption. Phagocytosis is the engulfing of foreign bodies by alveolar macrophages and their subsequent removal either up the ciliary "escalator" or by entrance into the lymphatic system. Some inhaled soluble particles are absorbed into the blood and translocated to other organs and tissues.

D.3.3 Internal Emitters

An internal emitter is a radionuclide that is inside the body. The absorbed dose from internally deposited radioisotopes depends on the energy absorbed per unit tissue by the irradiated tissue. For a radioisotope distributed uniformly throughout an infinitely large medium, the concentration of absorbed energy must be equal to the concentration of energy emitted by the isotope. An infinitely large medium may be approximated by a tissue mass whose dimensions exceed the range of the particle. All alpha and most beta radiation will be absorbed in the organ (or tissue) of reference. Gamma-emitting isotope emissions

are penetrating radiation, and a substantial fraction of gamma energy may be absorbed in tissue. The dose to an organ or tissue is a function of the effective retention half-time, the energy released in the tissue, the amount of radioactivity initially introduced, and the mass of the organ or tissue.

D.4 BIOLOGICAL EFFECTS OF RADIATION

When biological material is exposed to ionizing radiation, a chain of cellular events occurs as the ionizing particle passes through the biological material. A number of theories have been proposed to describe the interaction of radiation with biologically important molecules in cells and to explain the resulting damage to biological systems from those interactions. Many factors may modify the response of a living organism to a given dose of radiation. Factors related to the exposure include the dose rate, the energy of the radiation, and the temporal pattern of the exposure. Biological considerations include factors such as species, age, sex, and the portion of the body exposed. Several excellent reviews of the biological effects of radiation have been published, and the reader is referred to these for a more in-depth discussion (Brodsky 1996; Hobbs and McClellan 1986; ICRP 1984; Mettler and Moseley 1985; Rubin and Casarett 1968).

D.4.1 Radiation Effects at the Cellular Level

According to Mettler and Moseley (1985), at acute doses up to 10 rad (100 mGy), single strand breaks in DNA may be produced. These single strand breaks may be repaired rapidly. With doses in the range of 50–500 rad (0.5–5 Gy), irreparable double-stranded DNA breaks are likely, resulting in cellular reproductive death after one or more divisions of the irradiated parent cell. At large doses of radiation, usually greater than 500 rad (5 Gy), direct cell death before division (interphase death) may occur from the direct interaction of free-radicals with essentially cellular macromolecules. Morphological changes at the cellular level, the severity of which are dose-dependent, may also be observed.

The sensitivity of various cell types varies. According to the Bergonie-Tribondeau law, the sensitivity of cell lines is directly proportional to their mitotic rate and inversely proportional to the degree of differentiation (Mettler and Moseley 1985). Rubin and Casarett (1968) devised a classification system that categorized cells according to type, function, and mitotic activity. The categories range from the most sensitive type, "vegetative intermitotic cells, " found in the stem cells of the bone marrow and the gastrointestinal tract, to the least sensitive cell type, "fixed postmitotic cells," found in striated muscles or long-lived neural tissues.

Cellular changes may result in cell death, which if extensive, may produce irreversible damage to an organ or tissue or may result in the death of the individual. If the cell recovers, altered metabolism and function may still occur, which may be repaired or may result in the manifestation of clinical symptoms. These changes may also be expressed at a later time as tumors or cellular mutations, which may result in abnormal tissue.

D.4.2 Radiation Effects at the Organ Level

In most organs and tissues the injury and the underlying mechanism for that injury are complex and may involve a combination of events. The extent and severity of this tissue injury are dependent upon the radiosensitivity of the various cell types in that organ system. Rubin and Casarett (1968) describe and schematically display the events following radiation in several organ system types. These include: a rapid renewal system, such as the gastrointestinal mucosa; a slow renewal system, such as the pulmonary epithelium; and a nonrenewal system, such as neural or muscle tissue. In the rapid renewal system, organ injury results from the direct destruction of highly radiosensitive cells, such as the stem cells in the bone marrow. Injury may also result from constriction of the microcirculation and from edema and inflammation of the basement membrane, designated as the histohematic barrier (HHB), which may

progress to fibrosis. In slow renewal and nonrenewal systems, the radiation may have little effect on the parenchymal cells, but ultimate parenchymal atrophy and death over several months result from HHB fibrosis and occlusion of the microcirculation.

D.4.3 Low Level Radiation Effects

Cancer is the major latent harmful effect produced by ionizing radiation and the one that most people exposed to radiation are concerned about. The ability of alpha, beta, and gamma radiation to produce cancer in virtually every tissue and organ in laboratory animals has been well-demonstrated. The development of cancer is not an immediate effect. Radiation-induced leukemia has the shortest latent period at 2 years, while other radiation induced cancers have latent periods >20 years. The mechanism by which cancer is induced in living cells is complex and is a topic of intense study. Exposure to ionizing radiation can produce cancer at any site within the body; however, some sites appear to be more common than others, such as the breast, lung, stomach, and thyroid.

DNA is a major target molecule during exposure to ionizing radiation. Other macromolecules, such as lipids and proteins, are also at risk of damage when exposed to ionizing radiation. The genotoxicity of ionizing radiation is an area of intense study, as damage to the DNA is ultimately responsible for many of the adverse toxicological effects ascribed to ionizing radiation, including cancer. Damage to genetic material is basic to developmental or teratogenic effects, as well. However, for effects other than cancer, there is little evidence of human effects at low levels of exposure.

D.5 UNITS IN RADIATION PROTECTION AND REGULATION

D.5.1 Dose Equivalent and Dose Equivalent Rate

Dose equivalent or rem is a special radiation protection quantity that is used, for administrative and radiation safety purposes only, to express the absorbed dose in a manner which considers the difference in biological effectiveness of various kinds of ionizing radiation. The ICRU has defined the dose equivalent, H, as the product of the absorbed dose, D, and the quality factor, Q, at the point of interest in biological tissue. This relationship is expressed as $H = D \times Q$. The dose equivalent concept is applicable only to doses that are not great enough to produce biomedical effects.

The quality factor is a dimensionless quantity that depends in part on the stopping power for charged particles, and it accounts for the differences in biological effectiveness found among the types of radiation. Originally relative biological effectiveness (RBE) was used rather than Q to define the quantity, rem, which was of use in risk assessment. The generally accepted values for quality factors for various radiation types are provided in Table D-3. The dose equivalent rate is the time rate of change of the dose equivalent to organs and tissues and is expressed as rem/unit time or sievert/unit time.

Table D-3. Quality Factors (Q) and Absorbed Dose Equivalencies

Type of radiation	Quality factor (Q)	Absorbed dose equal to a unit dose equivalent*
X, gamma, or beta radiation	1	1
Alpha particles, multiple-charged particles, fission fragments and heavy particles of unknown charge	20	0.05
Neutrons of unknown energy	10	0.1
High-energy protons	10	0.1

*Absorbed dose in rad equal to 1 rem or the absorbed dose in gray equal to 1 sievert.

Source: USNRC. 1999. Standards for the protection against radiation, table 1004(b).1. 10 CFR 20.1004. U.S. Nuclear Regulatory Commission, Washington, D.C.

D.5.2 Relative Biological Effectiveness

RBE is used to denote the experimentally determined ratio of the absorbed dose from one radiation type to the absorbed dose of a reference radiation required to produce an identical biologic effect under the same conditions. Gamma rays from cobalt-60 and 200–250 keV x-rays have been used as reference standards. The term RBE has been widely used in experimental radiobiology, and the term quality factor used in calculations of dose equivalents for radiation safety purposes (ICRP 1977; NCRP 1971; UNSCEAR 1982). RBE applies only to a specific biological end point, in a specific exposure, under specific conditions to a specific species. There are no generally accepted values of RBE.

D.5.3 Effective Dose Equivalent and Effective Dose Equivalent Rate

The absorbed dose is usually defined as the mean absorbed dose within an organ or tissue. This represents a simplification of the actual problem. Normally when an individual ingests or inhales a radionuclide or is exposed to external radiation that enters the body (gamma), the dose is not uniform throughout the whole body. The simplifying assumption is that the detriment will be the same whether the body is uniformly or non-uniformly irradiated. In an attempt to compare detriment from absorbed dose of a limited portion of the body with the detriment from total body dose, the ICRP (1977) has derived a concept of effective dose equivalent. The effective dose equivalent, H_E, is

$$H_E = (\text{the sum of}) \; W_t \, H_t$$

where H_t is the dose equivalent in the tissue, W_t is the weighting factor, which represents the estimated proportion of the stochastic risk resulting from tissue, T, to the stochastic risk when the whole body is uniformly irradiated for occupational exposures under certain conditions (ICRP 1977). Weighting factors for selected tissues are listed in Table D-4.

The ICRU (1980), ICRP (1984), and NCRP (1985) now recommend that the rad, roentgen, curie, and rem be replaced by the SI units: gray (Gy), Coulomb per kilogram (C/kg), Becquerel (Bq), and sievert (Sv), respectively. The relationship between the customary units and the international system of units (SI) for radiological quantities is shown in Table D-5.

Table D-4. Weighting Factors for Calculating Effective Dose Equivalent for Selected Tissues

	Weighting factor		
Tissue	ICRP60	NCRP115/ ICRP60	NRC
Bladder	0.040	0.05	–
Bone marrow	0.143	0.12	0.12
Bone surface	0.009	0.01	0.03
Breast	0.050	0.05	0.15
Colon	0.141	0.12	–
Liver	0.022	0.05	–
Lung	0.111	0.12	0.12
Esophagus	0.034	0.05	–
Ovary	0.020	0.05	–
Skin	0.006	0.01	–
Stomach	0.139	0.12	–
Thyroid	0.021	0.05	0.03
Gonads	0.183	0.20	0.25
subtotal	0.919	0.99	0.70
Remainder	0.081	0.05	0.30

ICRP60 = International Commission on Radiological Protection, 1990 Recommendations of the ICRP;
NCRP115 = National Council on Radiation Protection and Measurements. 1993. Risk Estimates for Radiation Protection, Report 115. Bethesda, Maryland; NRC = Nuclear Regulatory Commission.
NRC = Nuclear Regulatory Commission, Title 10, Code of Federal Regulations, Part 20

Table D-5. Comparison of Common and SI Units for Radiation Quantities

Quantity	Customary units	Definition	SI units	Definition
Activity (A)	curie (Ci)	3.7×10^{10} transformations s^{-1}	becquerel (Bq)	s^{-1}
Absorbed dose (D)	rad (rad)	10^{-2} Jkg^{-1}	gray (Gy) Jkg^{-1}	
Absorbed dose rate (Š)	rad per second (rad s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	gray per second (Gy s^{-1})	Jkg^{-1} s^{-1}
Dose equivalent (H)	rem (rem)	10^{-2} Jkg^{-1}	sievert (Sv)	Jkg^{-1}
Dose equivalent rate ( )	rem per second (rem s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	sievert per second (Sv s^{-1})	Jkg^{-1} s^{-1}
Linear energy transfer (LET)	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}

Jkg^{-1} = Joules per kilogram; $Jkg^{-1}s^{-1}$ = Joules per kilogram per second; Jm^{-1} = Joules per meter; s^{-1} = per second

APPENDIX D

REFERENCES FOR APPENDIX D

ATSDR. 1990a. Toxicological profile for thorium. U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

ATSDR. 1990b. Toxicological profile for radium. U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

ATSDR. 1990c. Toxicological profile for radon. U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

ATSDR. 1999. Toxicological profile for uranium. U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

BEIR III. 1980. The effects on populations of exposure to low levels of ionizing radiation. Committee on the Biological Effects of Ionizing Radiations, National Research Council. Washington, DC: National Academy Press.

BEIR IV. 1988. Health risks of radon and other internally deposited alpha emitters. Committee on the Biological Effects of Ionizing Radiations, National Research Council. Washington, DC: National Academy Press.

BEIR V. 1988. Health effects of exposure to low levels of ionizing radiation. Committee on the Biological Effects of Ionizing Radiations, National Research Council. Washington, DC: National Academy Press.

Brodsky A. 1996. Review of radiation risks and uranium toxicity with application to decisions associated with decommissioning clean-up criteria. Hebron,Connecticut: RSA Publications.

Cember H. 1996. Introduction to health physics. New York., NY: McGraw Hill.

Early P, Razzak M, Sodee D. 1979. Nuclear medicine technology. 2nd ed. St. Louis: C.V. Mosby Company.

Eichholz G. 1982. Environmental aspects of nuclear power. Ann Arbor, MI: Ann Arbor Science.

Hendee W. 1973. Radioactive isotopes in biological research. New York, NY: John Wiley and Sons.

Hobbs C, McClellan R. 1986. Radiation and radioactive materials. In: Doull J, et al., eds. Casarett and Doull's Toxicology. 3rd ed. New York, NY: Macmillan Publishing Co., Inc., 497-530.

ICRP. 1977. International Commission on Radiological Protection. Recommendations of the International Commission on Radiological Protection. ICRP Publication 26. Vol 1. No. 3. Oxford: Pergamon Press.

ICRP. 1979. International Commission on Radiological Protection. Limits for intakes of radionuclides by workers. ICRP Publication 20. Vol. 3. No. 1-4. Oxford: Pergamon Press.

ICRP. 1979. Limits for Intakes of Radionuclides by Workers. Publication 30. International Commission on Radiological Protection. Pergamon Press.

ICRP. 1984. International Commission on Radiological Protection. A compilation of the major concepts and quantities in use by ICRP. ICRP Publication 42. Oxford: Pergamon Press.

ICRP. 1990. International Commission on Radiological Protection 1990 Recommendations of the ICRP

ICRU. 1980. International Commission on Radiation Units and Measurements. ICRU Report No. 33. Washington, DC.

James A. 1987. A reconsideration of cells at risk and other key factors in radon daughter dosimetry. In: Hopke P, ed. Radon and its decay products: Occurrence, properties and health effects. ACS Symposium Series 331. Washington, DC: American Chemical Society, 400-418.

James A, Roy M. 1987. Dosimetric lung models. In: Gerber G, et al., ed. Age-related factors in radionuclide metabolism and dosimetry. Boston: Martinus Nijhoff Publishers, 95-108.

Kondo S. 1993. Health effects of low-level radiation. Kinki University Press, Osaka, Japan (available from Medical Physics Publishing, Madison, Wisconsin).

Kato H, Schull W. 1982. Studies of the mortality of A-bomb survivors. Report 7 Part 8, Cancer mortality among atomic bomb survivors, 1950-78. Radiat Res 90;395-432.

Mettler F, Moseley R. 1985. Medical effects of ionizing radiation. New York: Grune and Stratton.

NCRP. 1971. Basic radiation protection criteria. National Council on Radiation Protection and Measurements. Report No. 39. Washington, DC.

NCRP. 1985. A handbook of radioactivity measurements procedures. 2nd ed. National Council on Radiation Protection and Measurements. Report No. 58. Bethesda, MD:

NCRP. 1993. Risk estimates for radiation protection. National Council on Radiation Protection and Measurements. Report 115. Bethesda, Maryland

Otake M, Schull W. 1984. Mental retardation in children exposed in utero to the atomic bombs: A reassessment. Technical Report RERF TR 1-83, Radiation Effects Research Foundation, Japan.

Rubin P, Casarett G. 1968. Clinical radiation pathology. Philadelphia: W.B. Sanders Company, 33.

UNSCEAR. 1977. United Nations Scientific Committee on the Effects of Atomic Radiation. Sources and effects of ionizing radiation. New York: United Nations.

UNSCEAR. 1982. United Nations Scientific Committee on the Effects of Atomic Radiation. Ionizing radiation: Sources and biological effects. New York: United Nations.

UNSCEAR. 1986. United Nations Scientific Committee on the Effects of Atomic Radiation. Genetic and somatic effects of ionizing radiation. New York: United Nations.

UNSCEAR. 1988. United Nations Scientific Committee on the Effects of Atomic Radiation. Sources, effects and risks of ionization radiation. New York: United Nations.

UNSCEAR. 1993. United Nations Scientific Committee on the Effects of Atomic Radiation. Sources and effects of ionizing radiation. New York: United Nations.

USNRC. 1999. Standards for the protection against radiation, table 1004(b).1. 10 CFR 20.1004. U.S. Nuclear Regulatory Commission, Washington, D.C.

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Two-Edged Sword

The owners' vote Tuesday was decisive: 28-2 in favor of eliminating two teams before the 2002 baseball season. Bud Selig did not name them, but the Expos and Twins are the likeliest to go. If a new labor battle is brewing, the first message has been sent, Sean McAdam writes.

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